THESIS

FOR THE AWARD OF DEGREE OF

MASTER OF SURGERY (General Surgery)

SESSION 2020-2023

"A CLINICOPATHOLOGICAL STUDY OF ULCERS ON THE LOWER LIMB AND ITS MANAGEMENT"

DR. SHAIK. NABI RUKSANA

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NO. 1, SANTOSH NAGAR,
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"I have had dreams and I have had nightmares, but I have conquered my nightmares because of my dreams"

-Dr. Jonas Salk

After completing my M.B.B.S in the year 2017, I waited almost 3 years before getting selected for post-graduation course in this institute. Being inspired by my father from a very young age, I have had a very specific dream about being a not only a doctor but to become a surgeon. That's why, when I got chance into Santosh Medical College and Hospital in my dream branch, the far distance, different language, an unknown environment and fully fledged COVID blizzard didn't deter me from coming here. Though the never-before-seen COVID situation made our life as junior residents, extremely difficult, it taught us a great deal about public health, treatment of critical patients, patient-doctor relationship, attenders counselling and hard work, empathy, compassion as in general.

I am deeply humbled and immensely grateful to my guide ***Prof. (Dr.) Gyanendra S Mittal*** *for the help, mentorship and valuable inputs, he offered to me and my colleagues regarding the thesis and during the entire tenure of post-graduation. I am grateful for his unvarying support, timely counsel, meticulous inspection, scientific approach and scholarly advice and guidance throughout my post-graduation which has helped me accomplish this research. He has always been approachable, understanding, calm and reasonable to our problems and with nippy and witty solutions for the same.*

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Dr. Shaik Nabi Ruksana

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INTRODUCTION

INTRODUCTION

An ulcer is defined as a disruption of the skin with erosion of the underlying subcutaneous tissue. This breach may extend further to the contiguous muscle and bone.

A chronic ulcer is defined as a full-thickness skin defect with no significant re-epithelization for more than 4 weeks or Ulceration can be defined as wounds with "full thickness depth" and a "slow healing tendency" in general the slow healing tendency is not simply explained by depth and size. But caused by an underlying pathologic fact that needs to be removed to induce healing.

Chronic ulceration of the lower leg and foot is frequent condition and wide in distribution they may be associated with a number of Medical, Surgical & Dermatological condition the patient suffering is very immense, commonly seen in most of the surgical wards and OPD.

The problems of leg ulcer represent a wide spectrum of etiology, pathology, severity and morbidity. The main causes are venous valve insufficient, lower extremity arterial disease and diabetes. Less frequent conditions are infections, vasculitis, skin malignancies and ulcerating skin diseases such as pyoderma gangrenosum.

But even rare condition exists such as recently discovered combination of vasculitis and hypercoagulability. For a proper treatment of patients with leg ulcers, it is important to be aware of the large differential diagnosis of leg ulceration. The causes may be various but the anatomical situation of ulcers in the leg by itself can give rise to problems that can at times test the ingenuity and patience of the surgeons.

The incident of ulceration is more in aging population and increased risk factor for atherosclerotic occlusion such as smoking, obesity and Diabetics. During the past three decades considerable knowledge has been gained regarding the physiology, anatomy, pathology and management of chronic leg ulcers. Despite all this the management of chronic leg ulcers is a fertile field for experimentation.

The article venous leg ulcers: Pathophysiology and classification by B Vasudevan [1] was interesting, and the author has dealt very eloquently with the subject of pathophysiology, classification, clinical assessment and investigatory approach to the patient with a leg ulcer. There are many causes for leg ulceration.

However, the majority (>90%) of chronic leg ulcers have a vascular etiology, with chronic venous hypertension thought to be the primary cause of around 70% and a significant contributory factor in a further 15% that has mixed arterial and venous etiological factors. Approximately, 5% occur due to arterial insufficiency. The rest may be due to diabetes, vasculitis, malignancy, hematological disorders, rheumatoid arthritis, pressure and other trauma. [2]

The pathophysiology of venous ulceration, including the clinical, etiologic, anatomical and pathophysiological (CEAP) classification, so important for clinical assessment and management, has been discussed in detail by the authors. A brief mention of pathophysiology of other common ulcers needs to be made. Arterial leg ulcers occur as a result of reduced arterial blood flow and subsequent tissue perfusion. Three mechanisms involved in the pathophysiology are (a) extramural strangulation, (b) mural thickening or accretion, and (c) intramural restriction of blood flow.

Diabetic foot ulcers are common and estimated to affect 15% of all diabetic individuals during their lifetime. The etiology of diabetic foot ulcers usually has many components. Peripheral neuropathy has been demonstrated to be the single most common contributing cause of foot ulceration. Ulceration can be attributed to a triad of peripheral neuropathy, biomechanical deformity, and superimposed minor trauma. [3] In general, lack of sensation from neuropathy is responsible for unrecognized repetitive trauma and loading that leads to skin and soft tissue breakdown, creating an entry point for infection.

Therefore, understanding the cause, presentation, and treatment of diabetic peripheral neuropathy is paramount for prevention of diabetic lower extremity ulcerative disease and amputation. To add to the woes, an inflammatory environment contributes to diabetic microangiopathy, worsening local ischemia.[4] Other factors in ulceration are trauma, deformity, callus formation, and edema. [5]

Pressure ulcers are as their name implies, caused primarily by unrelieved pressure. They usually occur over bony prominences such as the sacrum, trochanter or the heel but can occur on any part of the body subjected to pressure. They develop due to the compression and localized damage of skin and the underlying tissue between a bony prominence and an external surface.

A detailed history may provide clues to the etiology of the ulcer and should be confirmed by physical examination and appropriate investigations. Venous ulcers most commonly occur above the medial or lateral malleoli. Arterial ulcers often affect the toes or shin or occur over pressure points. Neuropathic ulcers tend to occur on the sole of the foot or over pressure points.

Ankle brachial pressure index is of immense value, as has been aptly brought out by the authors. The newer modalities of investigations include color duplex ultrasound scanning which has become the gold standard for evaluation of venous obstruction, but is also used to assess the location and extent of reflux in venous ulcers.

A lot of stress is often laid on wound swab cultures and immediate initiation of "appropriate" systemic antibiotics. The clinician should desist from such deleterious actions. Antibiotics are to be started only if the patient is febrile and exhibits overt signs of sepsis that may include leukocytosis and, in the peri, -operative period to cover against a bacteremia following debridement. A quantitative bacterial culture is more specific and should be performed once wound infection is suspected. This is performed by curetting or biopsying the bed of the ulcer.

The quantitative biopsy is the current favorite for assessing microbial pathogens within the wound. [6],[7] Quantitative biopsies containing <105 organisms/g of tissue are considered significant, and systemic antibiotic therapy should be then considered. Future applications in investigating the patient may include gene variant analysis in patients with venous leg ulcers. This implies that the high-risk minority of patients could be identified in advance by means of a simple blood test that could act as a genetic screening device. [8]

An ideal management plan for patients with leg ulcers should involve an early strategic and coordinated approach to delivering the correct treatment option for each individual patient, based on the accurate assessment of the underlying pathophysiology.

AIMS AND OBJECTIVES

AIM AND OBJECTIVES

Aim

To study the clinic pathological features of ulcers on the lower limb and its management.

Objectives

The main objectives of study are as follows:

1) To compare and analyze the distribution of age, sex, systemic diseases in the lower limb ulcers among 65 cases of the study group.
2) To study the clinical features of various types of lower limb ulcers.
3) To study the usefulness of applied investigations.
4) To effectively manage the conditions.
5) To prevent as far as possible leg ulceration in high-risk individual prone to the condition.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HISTORICAL ASPECT OF LEG ULCERS

The incidence of chronic leg ulcer is as old as history, as with any disease of mankind. The most common and noted ulceration of the chronic leg ulcers for many years is Stasis ulcers.

Hippocrates was the first person who apparently recognized some relationship between leg ulcers and venous disorders. A Roman physician Aurelius Cornelius celsus (25 BC – 50 AD) advised the use of plasters and linen bandages in the treatment of ulcers and also treated varicose veins by avulsion and cauterization. During the dark ages those with unsightly and ulcerated legs were denied simple remedies because it was believed that bandaging varicose legs would reintroduce black bile into the circulation and madness.

Richard Wiseman described a laced stocking in 1676. During the 18th century it began to be realized that leg ulcers were not necessarily accompanied by visible varicose veins. John Gay in 1868 described about the perforating veins of the calf and ankle, recorded the fact that ulcers could occur in the absence of varicose veins if there had been post thrombotic damage to deep veins and introduced the term 'Venous Ulcers". He described clot formation and post thrombotic recanalization. He correctly described the sub-fascial course of proximal short saphenous vein in upper 1/3rd of calf.

Homan divided ulcers into those associated with varicose veins of legs, easily cured by removal of these veins and post phlebitic ulcers, rapid in development, always refractory to palliative treatment not cured by varicose veins alone and needed excision. Home n in 1916 emphasized that sapheno -femoral ligation was a vital step in the prevention of the recurrence of varicosities.

Linton of Boston described about the sub fascial ligation of incompetent perforating veins. Sir Benjamin Collin Brodie (1789 - 1862) was the first to notice and describe the reverse flow of blood by means of a clinical test. Half a century later, this test was termed as "Trendelenburg's test".

Gay & Spencer's in 1868 wrote 2 important books on venous ulcers, which stressed the role of deep vein thrombosis and other lesions of arteries and veins (both superficial and deep) in etiology of leg ulcerations.

Gay also described perforating ankle veins and suggested use of the term venous ulceration. (M. Wayne Flye) [9] Linton drew attention to incompetence of communicating calf veins as potential cause of venous ulceration. (Linton RR) [10]

Avicenna (982 - 1027 AD) gave a good account of diabetes and was the first one to describe diabetic gangrene. Pryce (1887) described the association of ulceration and vascular diseases in diabetes.

In 1909 Burger described a syndrome of vascular occlusion in which arteries, veins, and nerves of the extremities were involved in extensive fibrosis that resulted in ulceration. Although a minor traumatic incident is often the immediate cause of the ulcer, the underlying pathology is usually vascular. Callam et al (1985) estimated that 70 percent of leg ulcers are venous in origin, 10 per cent are arterial and 10-15 per cent are of mixed arterial/venous origin. More unusual causes include malignancy, vasculitis, neuropathy, metabolic disorders such as pyoderma gangrenosum, and ulcers associated with disorder such as sickle cell ulceration (Moffatt and Harper 1997).

INCIDENCE AND OCCURRENCE OF LEG ULCERS

Accurate data concerning the incidence of non-fatal diseases are difficult to obtain and statistics are usually derived from hospital attendance records and general practice surveys. The prevalence of a condition within a community may be affected by many factors such as the genetic constitution of its members, nutritional status, environmental factors, customs and occupation. In under developed countries, the incidence of a condition may be greatly under estimated and apparent differences between populations may be affected by differences in age structures.

The prevalence of leg ulcers is probably between 0.18% and 1% of the population. (Phillips Tania et al)[11] The site of ulceration is recorded using the method of Callum; 90% of the ulcers were present in the gaiter area, 2 % in the foot and 8% in the leg. (Baker S. R. et al) [12]

Incidence of ulcers is more common among women with no difference in age related prevalence. A basic study of 4422 healthy working adults aged between 20 and 70 years in Europe resulted in detection of chronic venous insufficiency in 19% of men and 25% of women. Other surveys conducted in Europe and America came out with similar results and surveys confirmed the increased incidence of ulcerations in females. Information from other parts of the world other than Europe and America is sparse, but it is generally held that ulcerations due to venous insufficiency are less common in African natives.

Schulz, Finaly and Scott (1962), in Transvaal, found that 0.1% of 2000 consecutive Bantu patients attending surgical outpatient department had venous ulcers, but do not give figures for the incidence of non-venous leg ulcers, while Dogliotti (1970) in Johannesburg found the incidence of leg ulcers (unspecified) in Bantu patients referred to hospital to be 1.75%.

Finally, and Park (1969) compared the incidence of varicose eczema in their middle-aged Indian patients (7%) with that in comparable "white" patients (3%) and Bantu (1%) but give no figures for the incidence of venous ulcerations. In Nigeria, Shrank and Harman (1966) found no varicose ulcers, 1.1% among 1156 Nigerian outpatients was noted to have tropical ulcers.

ANATOMY OF THE LEG AND FOOT

The leg is that part of the lower limb below the knee and the terminal part of the leg below the ankle is the foot.

Front of the leg and dorsum of foot

Superficial fascia of the front of the leg and the dorsum of the foot contains the following parts:

1. Superficial veins.
2. Cutaneous Nerves
3. Lymphatics
4. Small unnamed arteries.

The subcutaneous bony surfaces are not covered by the deep fascia of the leg but are attached to it at its borders. It is thick in the upper part of the leg and gives origin to the underlying muscles while in the lower part is thin and forms retinacula around the ankle. Deep fascia is extremely thin on the dorsum of the foot.

Intermuscular septa are formed in the deep fascia, which tends to divide the leg into compartments. The anterior and the posterior intermuscular septa are attached anterior and posterior borders of the fibula and divide the leg into anterior, lateral and posterior compartments. In the posterior compartment a superficial transverse facial septum separate tibialis posterior from the long flexors of the toe. Muscles of the anterior compartment are – Tibialis anterior, Extensor hallucis longus, Extensor digitorum longus and Peroneus tertius.

Anterior Tibial Artery

The main artery that is found in the anterior compartment of the leg is the anterior tibial artery. The perforating branch of the peroneal artery feeds this artery. Anterior tibial artery is the smaller terminal branch of popliteal artery.

Surface Anatomy

In surface projection of the anterior tibial artery begins 2.5 cm below the medial side of fibula and ends at the midpoint between the malleoli. The vessel can be felt pulsating lateral to the tendon of extensor hallucis longus at the ankle.

Course

The originating point of the anterior tibial artery is at the back of the leg at the lower border of the popliteus muscle and it penetrates the anterior compartment of the leg through an opening in the upper part of the interosseous membrane.

In the anterior compartment it runs vertically downwards to a point midway between the two malleoli, where it ends by becoming continuous as the dorsalis pedis artery. Anterior tibial artery is accompanied by two venae comitantes and the deep peroneal nerve is lateral to it in the upper and lower third and anterior to it in the middle 1/3rd of the leg.

Variations

The perforating branches of the peroneal artery and the perforating branches of the posterior tibial artery may partially or completely replace the anterior tibial artery.

Branches

The adjacent muscles are supplied by the muscular branches. Anastomotic branches feed the knee and the ankle. Anterior and posterior tibial recurrent branches take part in the anastomosis around the knee joint. Anterior medial malleolar and anterior lateral malleolar branches take part in the anastomosis around the ankle joint.

Medial malleolar network lies just below the medial malleolus. It is formed by:

1. Anterior medial malleolar branch of anterior tibial.
2. Medial tarsal branches of dorsalis pedis.
3. Medial malleolar branches of posterior tibial.
4. Calcaneus branches of posterior tibial and
5. Twigs from the lateral plantar artery

Lateral malleolar network lies just below the lateral malleolus. It is formed by:

1. Anterior lateral malleolar branch of anterior tibial.
2. Lateral branches of dorsalis pedis.
3. Perforating branch of peroneal artery.
4. Twigs from the lateral plantar artery.

Dorsalis Pedis Artery

The direct continuum of the anterior tibial artery, which forms the chief artery of the dorsum of the foot, is the dorsalis pedis artery.

Surface anatomy

Being superficial the vessel can be felt pulsating along a line from the midpoint between the malleoli to the proximal end of the first intermetatarsal space.

Course

The originating point of this artery is in front of the ankle between the two malleoli. The artery lies along the medial side of the dorsum of the foot and reaches the proximal end of the first intermetatarsal space. Its deep downwards between the two heads of the first dorsal interosseous muscle and ends in the side by completing the plantar arterial arch.

Relations

Superficial:

- Skin and fascia with interior and exterior retinaculum
- Extensor hallucis brevis, which crosses the artery from lateral to medial side

Deep:

- Capsular ligament of ankle joint
- Talus, navicular and intermediate cuneiform

Medial: Extensor hallucis longus

Lateral:

- First tendon of extensor digitorum longus
- Medial terminal branch of deep peroneal artery.

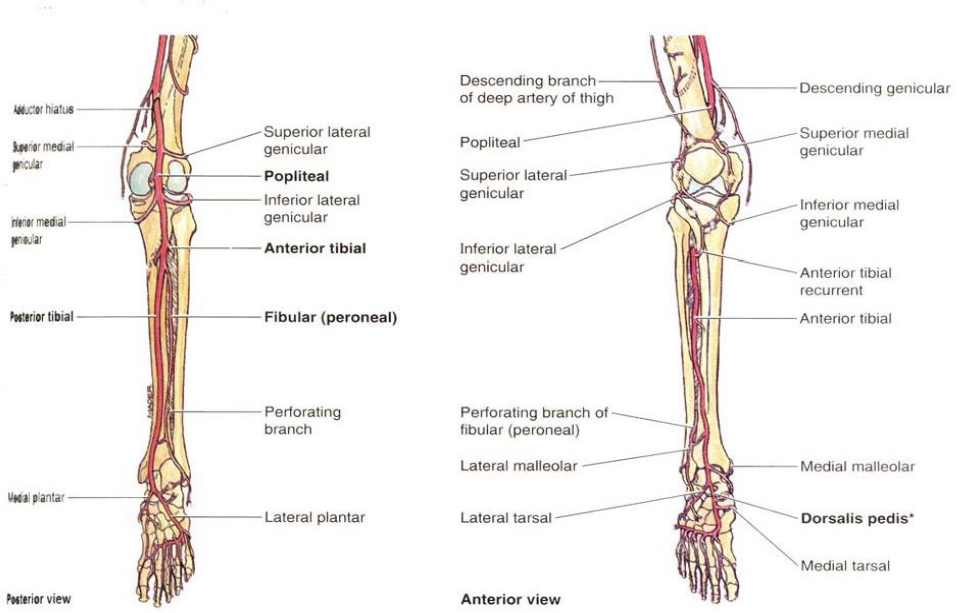


Figure 1: (A) Posterior tibial, popliteal, and lateral plantar artery. (B) Anterior tibial and dorsal pedis artery

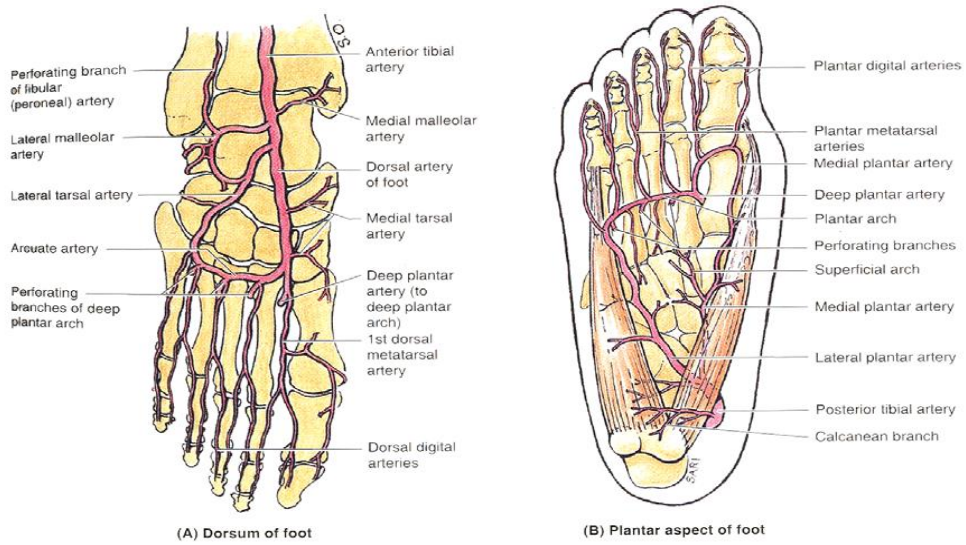


Figure 2:Arteries of the foot

Branches

- Lateral tarsal artery supplies extensor digitorum brevis, tarsal joints and ends in lateral malleolar network.
- Medial tarsal branches joint medial malleolar network.
- Arcuate artery is a large branch that arises opposite the medial cuneiform bone. It gives off second, third and fourth dorsal metatarsal artery each of which divides into dorsal digital branches for the adjoining toes.
- First dorsal metatarsal artery gives a branch to medial side of big toe and divides digital branches for adjacent sides of first and second toes.

Cutaneous Innervation

Saphenous nerve which is a branch of the posterior division of femoral nerve pierces the deep fascia the medial side of the knee, runs downwards in front of great saphenous vein and supplies skin of the medial side of the leg and medial border of the foot up to the ball of big toe.

Infrapatellar branch of saphenous nerve pierces the sartorius and deep fascia on the medial side of the knee and supplies the skin over the ligamentum patellae. Lateral cutaneous nerve of calf, which is a branch of the common peroneal nerve, supplies skin of their upper 2/3rd of lateral side of leg. Superficial peroneal nerve is a branch of common peroneal nerve on the lateral side of the neck of fibula. It pierces the deep fascia at the junction of the upper 2/3rd and the lower 1/3rd of the lateral side of the leg and supplies skin over the lower 1/3rd of the lateral side of the leg and the dorsum of the foot.

Sural nerve is a branch of the tibial nerve given of in the popliteal fossa. It pierces the deep fascia in the middle of the back of the leg accompanies the small saphenous vein and supplies the skin of the lower ½ of the back of the leg and lateral border of the foot up to the tip of the little toe. Deep peroneal nerve supplies the cleft and adjacent borders of the first and second toe. Digital branches of the medial and lateral plantar nerve supply the distal parts of the toes.

Deep Peroneal Nerve

This is the nerve of the anterior compartment of the leg and the dorsum of the foot. It is one of the two terminal branches of peroneal nerve.

Course. Originating on the lateral side of the neck of the fibula, it enters the anterior compartment by piercing through the anterior intermuscular septum from where it accompanies the anterior tibial artery in to the leg. It is the main supplier to the anterior muscle compartment.

The nerve ends on the dorsum of the foot close to the ankle joint by dividing into lateral and medial terminal branches. The extensor digitorum brevis, tarsal joints and the second dorsal interosseous muscles are supplied by the lateral terminal branch. The medial terminal branch ends by supplying the first interdigital cleft, proximal joint of the big toe and often the first dorsal interosseous muscle.

Branches The adjacent sides of the first and the second toes are supplied by the cutaneous branches. The muscles of the anterior compartment of the leg and the extensor digitorum brevis on the dorsum of the foot are supplied to by the muscular branches. Articular branches supply the ankle joint, tarsal joint, tarsometatarsal and metatarsophalangeal joint of the big toes.

Lateral Side of the Leg

The lateral compartment of the leg is a bound unit. There are many muscular members involved in binding this compartment, they are:

1. Anteriorly by anterior intramuscular septum
2. Posteriorly by posterior intramuscular septum
3. Medially by lateral surface of fibula.
4. Laterally by deep fascia of the leg.

Contents:

1. Muscles
2. Peroneus longus
3. Peroneus brevis

Actions

The peroneus longus and the brevis are evertors of the foot. The movements of inversion and eversion help to adapt the foot to uneven ground. Along with the other muscles present around the ankle the peroneal help to maintain the stability of the joint. The peroneus longus helps to maintain the lateral longitudinal arch of the foot.

Vessels

Arterial supply is derived from the branches of peroneal artery; veins drain into small saphenous vein.

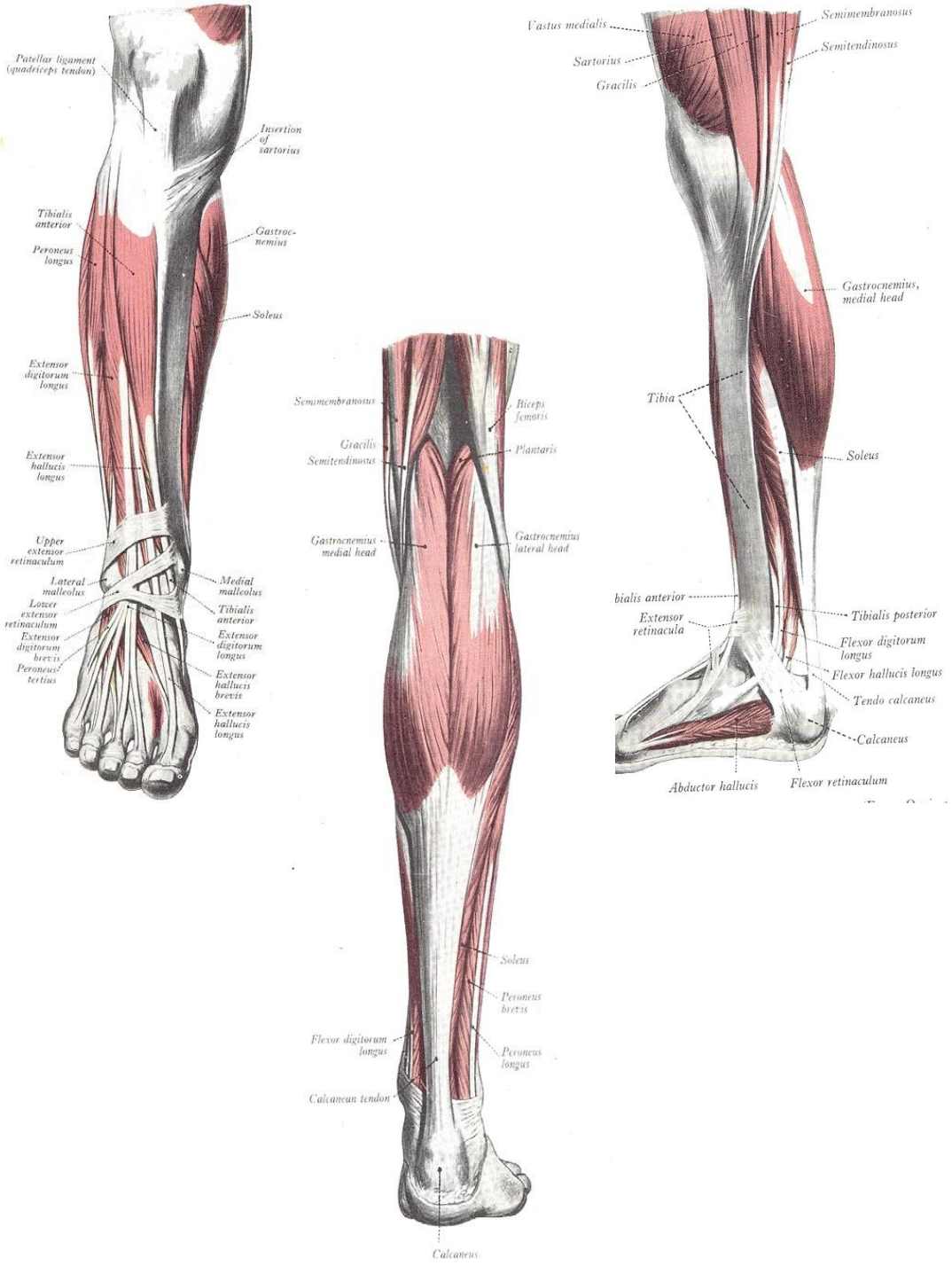


Figure 3: Muscles of the leg

Nerve

1. Superficial peroneal nerve
2. Superficial Peroneal Nerve
3. One of the common peroneal nerve's two terminal branches which is the main nerve to the lateral compartment of the leg is the superficial peroneal nerve.

Course and Relation

It begins on the lateral side of the neck fibula, descends through the substance of peroneus longus in the upper 1/3rd, lies between the peroneus longus and brevis muscle in the middle 1/3rd and then descends to a groove between the peroneus brevis and extensor digitorum longus. At the junction of the upper 2/3rd and lower 1/3rd of the leg it pierces deep fascia and divides into medial and lateral branches, which descend into the foot.

Branches and distribution

1. Muscular branches – To peroneus longus and brevis.
2. Cutaneous branches – through the terminal branches supplies 1/3 of the lateral side of the leg and the dorsum of the foot. The lateral branch supplies the third and the fourth clefts of the toes.
3. Communication branches – communication between the saphenous and the deep peroneal nerves takes through the medial branch and the lateral branch with the sural nerve.

Medial side of the leg

The medial surface of the shaft of the tibia is the originator of this part of the leg. A large part of the medial surface of the leg is covered only by the skin and the superficial fascia and hence this part is largely subcutaneous.

The surface at the upper end provides anchorage to the tibial collateral ligament and also acts as the insertion point of the sartorius, gracilis and semitendinosus, all of which are covered by a thin layer of deep fascia. The great saphenous vein lies in the superficial fascia as they cross the lower 1/3 of this surface. This skin, fascia and the periosteum of this surface are supplied by saphenous nerve.

Rear of the leg

The hind leg contains the posterior compartment of the leg. This posterior compartment is bound by the following:

Anteriorly by the rear surface of the:

1. Tibia
2. Interosseous membrane
3. Fibula
4. Posterior intermuscular septum

Posteriorly by the deep fascia of the leg. Superficial fascia of the back of the leg.

Contains:

1. The small and the great saphenous vein
2. Several cutaneous nerve
3. Medial and lateral calcaneal arteries.

The muscles of the posterior compartment are divided into superficial and deep Group. The superficial muscles are the gastrocnemius, Soleus and the Plantaris. The deep muscles are the Popliteus, Flexor digitorum longus, Flexor hallucis longus and the Tibialis posterior.

Posterior Tibial Artery

This is the larger terminal branch of the popliteal artery.

Surface anatomy

The posterior tibial artery from the middle line of the calf at the level of neck of fibula to the midpoint between the medial malleolus and the prominence of the heal, where its pulsations can be felt.

Course

Originating at the lower border of the popliteus between the tibia and the fibula deep to gastrocnemius, it enters the back of the leg and passes deep into the soleus. It then runs downwards and medially to reach midway between the medial malleolus and medial tubercle of calcaneum to terminate deep into the flexor retinaculum by dividing into a large lateral and a small medial plantar artery.

Branch:

1. Peroneal artery
2. Muscular branches for the posterior compartment.
3. Nutrient artery to tibia is the largest nutrient artery in the body.
4. Anastomotic branches – Circumflex fibular for the knee, communicating branch for peroneal, medial malleolar and calcaneal artery for ankle and heel.
5. Terminal branches – medial and lateral plantar.

Peroneal Artery

The lateral and the posterior compartments of the leg are supplied by the largest branch of the posterior tibial artery and are called the peroneal artery.

Course

Beginning just 2.5 cm below the lower border of the popliteus, it descends along the medial crest of the fibula. It is found in a fibrous canal between the tibialis and the flexor hallucis longus, it further continues downward behind the ankle joint and terminates into the lateral calcaneum branches.

Branches

1. Muscular branches to posterior and lateral branches.
2. Nutrient artery to the fibula.
3. Anastomotic branches.
4. Perforating branch joins the lateral malleolar network.
5. Communicating branch anastomosis with a similar branch from posterior tibia.
6. Calcaneus branches join the lateral malleolar network.

Cutaneous Innervation

Saphenous nerve is a branch of posterior division of femoral nerve and supplies the skin of medial side of the leg and medial border of foot up to the ball of the big toe. Posterior division of the medial cutaneous nerve of thigh supplies the skin of the uppermost parts of medial area of the calf. Posterior cutaneous nerve of thigh supplies the skin of the upper ½ of the central area of the calf muscle. Sural nerve is a branch of the tibial nerve and supplies the skin of the lower half the central area of the leg, lower 1/3rd of the lateral area of the calf and also the lateral border of the foot up to the tip of the little toe. Lateral cutaneous nerve of calf is branch of the common peroneal nerve and supplies the skin of the upper 2/3rd of the lateral area of leg both in front and behind.

Sole of the foot

The skin of the sole of the foot is thick, firmly adherent to the underlying plantar aponeurosis and is creased.

Cutaneous innervations

Medial calcaneal branches of tibial nerve supply the skin of posterior and medial portions of the sole. Branches from medial plantar nerve of the anteromedial portions with medial 3 ½ digits. Branches from lateral plantar nerve supplies the anterolateral portions with 1 ½ lateral digits.

Superficial fascia

This is more fibrous and denser. The fibrous brands bind the skin to the deep fascia and divide the subcutaneous fat into small tight compartments. The fascia is very thick and dense over the weight bearing points.

1. Deep Fascia
2. Deep fascia of the sole forms:
3. Plantar aponeurosis in the sole.
4. Deep transverse metatarsal ligaments between the metatarsophalangeal joints.
5. Fibrous flexor sheaths in the toes.

Muscles of the first layer of the sole are Flexor digitorum brevis, Abductor hallucis and Abductor digitorum. The muscles of second layer are Flexor digitorum accessories and lumbricals, which are four in number. Muscles of third layer of sole are Flexor hallucis brevis, Adductor hallucis. and Flexor digiti minimum brevis. The fourth layer of the sole contains the muscles – the Dorsal interossei which are four in number and lie between metatarsal bones and the Plantar interossei which are three in number and lie below the metatarsal bones. The main arteries of the sole are the medial plantar artery and the lateral plantar artery. The main nerves of the sole are medial and lateral plantar nerves.

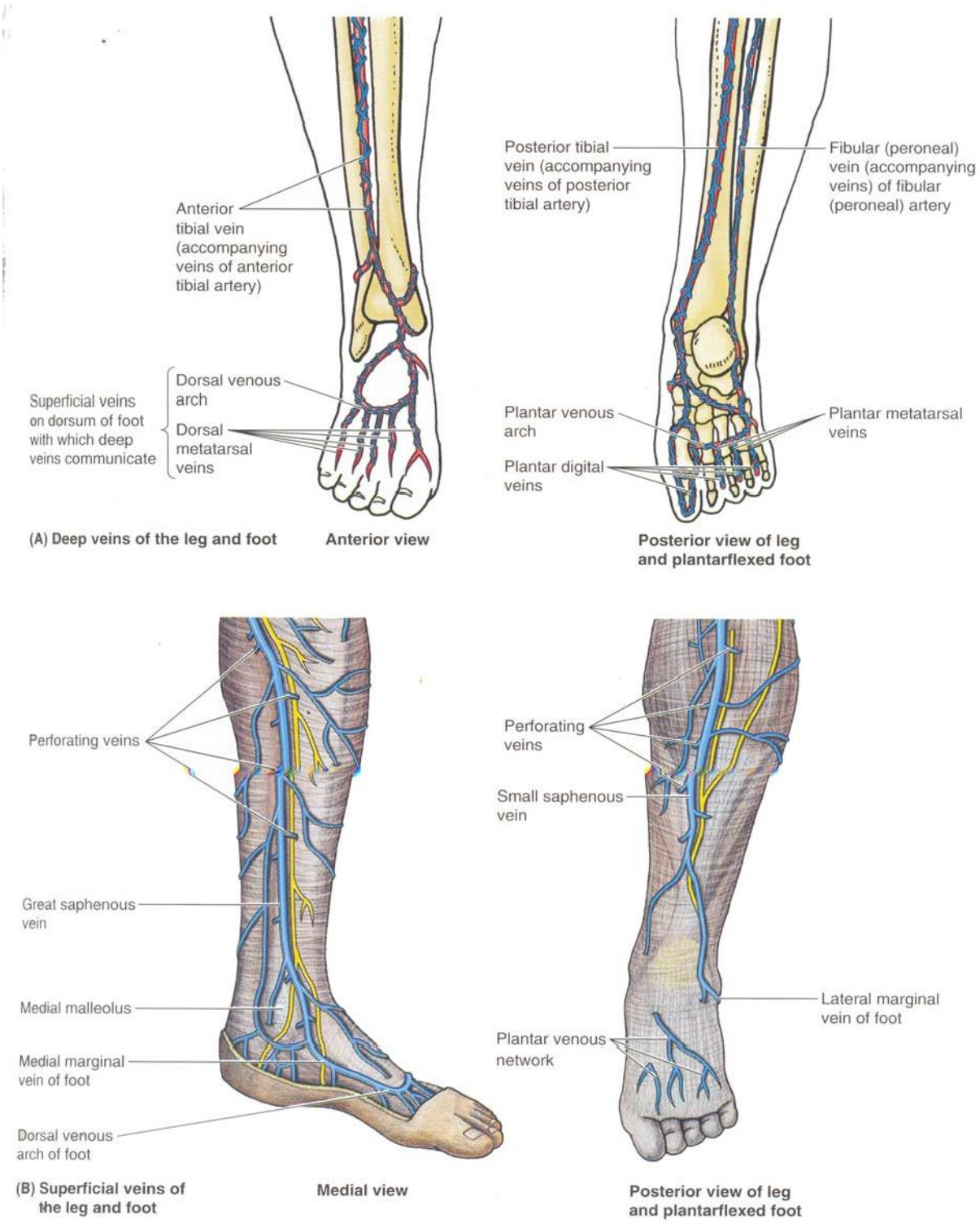

Anterior tibial vein (accompanying veins of anterior tibial artery)
Superficial veins on dorsum of foot with which deep veins communicate
Dorsal venous arch
Dorsal metatarsal veins
(A) Deep veins of the leg and foot
Anterior view
Posterior tibial vein (accompanying veins of posterior tibial artery)
Fibular (peroneal) vein (accompanying veins) of fibular (peroneal) artery
Plantar venous arch
Plantar digital veins
Plantar metatarsal veins
Posterior view of leg and plantarflexed foot
Perforating veins
Great saphenous vein
Medial malleolus
Medial marginal vein of foot
Dorsal venous arch of foot
(B) Superficial veins of the leg and foot
Medial view
Perforating veins
Small saphenous vein
Lateral marginal vein of foot
Plantar venous network
Posterior view of leg and plantarflexed foot

Figure 4: Superficial and deep veins of the leg and foot

Venous Drainage of Lower limb

The veins of lower limb may be divided into 3 groups.

1. Superficial
2. Deep
3. Perforating

Superficial Venous System

It consists of Great saphenous vein and small saphenous vein and their tributaries. These are situated beneath the skin in the superficial fascia. They are thick walled and valves are more numerous more distally. A large portion of their blood is drained into deep veins through the perforating veins.

Long/ Great Saphenous Vein

It begins on the dorsum of the foot from the medial end of the dorsal venous arch and runs upwards in front of the medial malleolus, along the medial side of the leg and behind the knee. In the thigh it opens into the femoral vein by piercing the cribriform fascia (4 cm below and lateral to the pubic tubercle). The junction of long saphenous vein and femoral vein is clearly visible as white line. Saphenous nerve is closely applied to the long saphenous vein in the leg and ankle where it is at risk of damage by vein stripping. This vein consists 10-15 valves which prevent backflow of venous blood encouraged by gravity. One valve is always present at the saphenofemoral junction. The vein is also connected to the deep veins by the perforating veins. Before piercing the cribriform fascia, it receives 3 named tributaries:

1. Superficial epigastric
2. Superficial external pudendal
3. Superficial circumflex iliac

Small Saphenous Vein

It is formed by the union of the lateral end of the dorsal venous arch with lateral marginal vein. It enters the back of the leg by passing behind the lateral malleolus. In the leg it ascends first lateral to tendocalcaneous and then along the middle line of the calf to the lower part of the popliteal fossa where it pierces the popliteal fossa and opens into the popliteal vein. It drains the lateral border of the foot, the heel and the back of the leg. It is connected with the great saphenous vein and with the deep veins and is accompanied by the Sural nerve.

Deep Veins

These are anterior and posterior tibial, peroneal, popliteal, femoral and their tributaries. They accompany the named arteries and are supported by the powerful surrounding muscles. The valves are more numerous than in superficial veins. The muscular veins are also valved except for the soleal veins. They are more efficient channels than the superficial veins because of the driving force of the muscular contraction.

Perforating Veins

These are communicating veins between superficial and deep veins.

1) INDIRECT

These are not important and are 50 to 100 in number. These consist of small superficial vessels which penetrate the deep fascia to connect with a vessel in a muscle which in turn is connected to one of the deep veins. Ankle is having little muscle; therefore, it has few indirect perforators. In ankle return of blood from superficial tissues depends only on direct perforators. This is the reason for ankle ulceration.

2) DIRECT

These are more important and consist of one long saphenous, two small saphenous and three perforating veins. Small and long saphenous veins are also considered as large direct perforators because they are valved to prevent flow from superficial to deep venous system.

Perforating Veins

They are fairly constant in position and related intimately to the posterior border of tibia and fibula and are valved. As a rule, they are accompanied by an artery.

1.Thigh Perforator (Adductor canal perforator): It communicates between long saphenous vein and femoral vein.

2.Leg Perforators:

A. Below Knee: Communicating vein between long saphenous or posterior arched vein and posterior tibial vein.

B. Medial Ankle Perforators: They connect with a serious of venous arcades which characterize the lower part of the large constant posterior arch vein.

i) Upper Perforator: Found at the junction of middle and lower third of leg at the posteromedial border of tibia. It constantly communicates with long saphenous vein. It is three to four centimeters long.

ii) Middle Perforator: It lies 10 cm above the tip of medial malleolus and just behind the tibial margin. It is 3-4 cms long.

iii) Lowest Perforator: It is situated behind and below the medial malleolus and is 1 cm long.

C. Lateral Perforator: A constant lateral perforating vein is found at the junction of lower and middle third on the outer side of leg. It connects small saphenous vein with peroneal vein.

Physiology Of Venous Flow in Lower Limb

Veins contain two thirds of total circulating blood volume. Venous tone is mediated by sympathetic system. The most important function of superficial veins of lower limb is thermo regulation. Deep veins mainly act as passive blood conduits. Blood is returned to heart by means of four pumps namely:

1. Foot Pump.
2. Abdominal Pump
3. Respiratory Pump.
4. Calf Pump.

Foot Pump: Blood is expelled from plantar veins by their intermittent stretching during foot movements, rather than by pressure of the soul of the foot on the ground.

Respiratory Pump: Shallow inspiration increases the total venous return slightly. But deep inspiration almost certainly reduces venous return.

Calf Pump (Peripheral heart): Disordered function of calf pump is the single most important factor in the etiology of venous ulceration. Calf muscle pump has four important components

1. Dilated valveless sinusoids within the soleus and gastrocnemius.
2. Direct perforating veins.
3. Numerous valves in the communicating and deep veins.
4. A layer of tough deep fascia which surrounds the calf muscles.

Although it has been suggested that all communicating veins (the long and short saphenous veins as well as the lower calf perforating veins) are subjected to high venous pressure in the presence of deep venous incompetence and reflux, when deep vein valves are incompetent, the pressures inserted on the lower calf and ankle perforating veins are significantly higher than those of either the long or short saphenous veins.

Resting foot venous pressure reflects the height between the right atrium and foot and in erect position is normal 80 – 90mm Hg. Following exercise, by raising the heel repeatedly off the ground, the pressure falls to about 25 mm Hg. Standing motionless, the foot venous pressure takes 25- 30 seconds to return to previous high resting levels, but venous reflux results in a rapid return to resting pressure following exercise.

Calf muscle pump function may become disordered as a result of muscle weakness, saphenous incompetence, deep vein incompetence or obstruction or perforating vein incompetence with or without deep vein incompetence. All these abnormalities will adversely affect pressure profiles in the dorsal foot vein. Most venous ulcers occur in the malleolar regions which are drained by the corona phlebectasia, the network of small veins communicating with the calf and the ankle perforating veins.

At present measurement of calf muscle pump function can be done by dorsal foot venous pressure measurement or by plethysmography. Measurement of flow and pressure changes in the ankle flare veins is extremely difficult but, it is the disorders of this venous network that are of prime importance in the etiology of venous ulcerations. In the prevention of recurrent alteration, all points of venous reflux communicating with the malleolar venous network must be accurately identified and effectively controlled.

LYMPHATIC DRAINAGE

Superficial lymphatics

These form large trunks and are more numerous than the deep lymphatics. They run in the superficial fascia in two streams.

The main stream follows the long saphenous vein and ends in the lower vertical group of the superficial inguinal lymph nodes.

The accessory stream follows the short saphenous vein and ends in the popliteal lymph nodes.

Deep Lymphatics

These are fewer than superficial lymphatics and drain all structures lying deep to the deep fascia. They terminate mostly into the deep inguinal nodes either directly or indirectly through popliteal nodes.

Figure 5: Layers of the skin

Stratum germinativum

Stratum spinosum

Stratum granulosum

Stratum lucidum

Stratum corneum

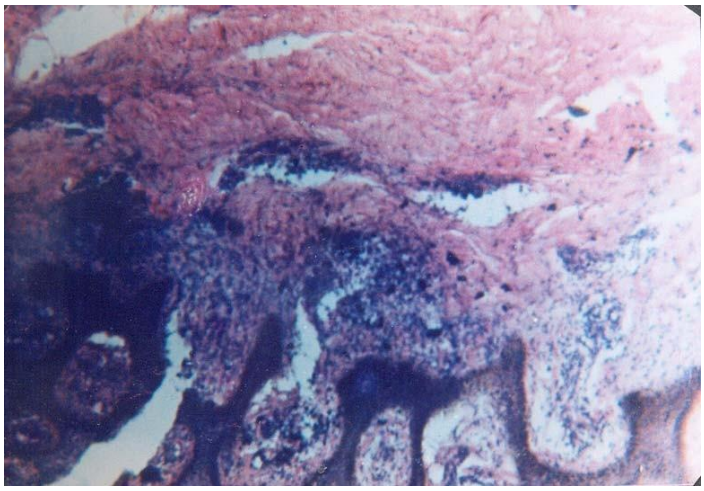


Figure 6: Squamous cell carcinoma – High power Field

HISTOLOGY OF THE SKIN

The skin is anatomically composed of three distinct strata and they are named as: (From the outermost to the inner layer).

1. Epidermis
2. Dermis
3. Subcutaneous fat.

The Skin

Its development - The ectoderm and the mesoderm act as the originator of all the constituents of the skin. Or to put it in simple terms the skin is developmentally derived from the ectoderm and the mesoderm.

The Epidermis

The epidermis is composed mostly of keratinocytes, and stratified squamous epithelium. The epidermis is made – up of the five layers.

The stratum corneum is the first layer and this is thickest in palmar and plantar areas. The second layer is stratum lucidum which is an even, colorless, translucent and a shining band just below stratum corneum. The third layer is stratum granulosum. The fourth layer is stratum malphighi (Stratum spinosum) which consists of row of mosaic like keratinocytes. The last and the fifth layer of the epidermis is the stratum germinativum (Basal layer). This layer consists of rectangular columnar cells arranged vertically over a wavy basement line.

Cell differentiation in Keratinocytes takes place in the direction of the external environment and they yield a cornified surface membrane. The process of differentiation whereby germinative keratinocytes move outward to become fully cornified cells takes approximately 14 days and are present in transit through the cornified layer. The replacement of the epidermis and in very thick epidermis, in the second layer as well.

In the basal layer 3-5 % of the cells synthesize DNA at any given time. During upward migration towards maturation, keratinocytes undergo characteristics changes. Basal, spinous, granular and cornified layers are the strata that reflect stages in the conversion of a germinative keratinocyte from the beginning to the end and produce the epidermal differentiation. In the upper Malpighian layer dendritic cells resembling monocytes are found and are called as Langerhans cells.

Melanin pigment is synthesized and secreted by melanocytes that are dendritic cells. The melanocytes are present all over the body but their distribution is not even. The intensity of coloration is further determined by the total number of cells and intercellular distribution of melanin granules within the epidermal keratinocytes.

The Dermis

The dermis or the corium is a dense fibrous or elastic layer, which provides the strength and elasticity to the skin. Composed primarily of white fibrous tissue, it also has some yellow elastic fibers and cement like substances called the cellular elements.

Blood vessels, glandular structures, lymph vessels, hair follicles, muscle elements prolongations of fatty tissue and nerves with terminal organs of touch and sensation are all contained and supported in this layer. The dermis is covered over its entire surfaces with numerous conical papillae. This stratum is called 'The papillary layer', whereas the deep stratum is termed 'The reticular layers. The combined anatomical unit of papillary and reticular dermis is called 'The adventitial dermises.

The level of intimacy between the papillary dermis and the epidermis is easily visible in their alterations as a result of inflammatory skin diseases. They form a morphological and a functional unit, which work together. Extending above the subcutaneous fat are thick collagen bundles, which are mostly arranged parallel to the skin surface as in orthogonal wicker work, and this layer is the composite matter of the dermis reticular layer.

Subcutaneous Fat

Subcutaneous fat, like the dermis is derived from mesenchyme and mesenchymal cells. These cells give rise to lipocytes (fat cells) as well as fibrocytes. The lipocytic cells manufacture so much fat inside themselves that in the process they tend to flatten the nucleus against the wall of the cell. Strands of collagen divide the population of the fat cells into lobules. These fibrous elements known as trabeculae have major vascular networks, lymphatics and nerves and are analogous to the adventitial dermis. The subcutaneous fat varies in thickness from one are to another. Functionally the subcutaneous fat acts as an insulator., shock absorber and a chronic reserve depot.

Nerves

Sensations such as touch, pressure, temperature, pain and itch are received by millions of microscopic dermal nerve endings. They are most numerous on hairless parts like palms, soles of the feet, fingers and clitoris, the erogenous zones. These tiny end organs terminate principally in the papillary dermis and around hair follicles. These nerve endings can be free or encapsulated, myelinated or unmyelinated and most of these are visualized with difficulty using the hematoxylin and eosin stain.

Healing by first intension

The healing of wounds caused by accident, assault, warfare and surgical operations has always been a central, consideration in surgical practice because any breach in the surface of the body – the skin and mucous membrane – exposes the deeper tissue to the danger of infection.

Therefore, it is necessary to assist the healing process of the body to restore an intact surface as soon as possible. Immediate closure of a wound (Primary suture) using sutures, clips and adhesive materials favor healing with minimal scarring and is called healing by first intension.

Healing by second intension

When the wound edges do not come together or when there is irreparable skin loss or when the wound becomes infected and breaks apart and has to be laid open, then in such cases healing takes place by second intension. In this type of healing, the wound is healed by a filling of granulation tissue. This process of healing is far slower than wound healing by first intension and there is more amount of scar tissue left behind (Ulcers usually heal in a similar fashion). Other methods of providing skin cover – in the presence of devitalized tissue, swelling tension and skin loss – include delayed primary suture, skin grafting and second suture.

Factors contributing to rate of wound healing

There are many factors, which govern the rate at which a wound heals. Some of these factors are discussed below:

Blood Supply

Wounds incorporated on the face and hands may seem horrifying initially but they tend to heal well because of good blood supply to these organs. Injuries sustained in relatively poorly supplied parts of the body like the areas below the knees, over the shin tend to take a much long time to heal.

Tension

Tension of the tissue inhibits the local blood supply and leads to wound failure. Local swelling and therefore tension builds up automatically during the first 48 hours after the injury as part of the phenomenon of inflammation. Hematoma, venous stasis (e.g., In a dependent limb) and infection also increase tension.

Age

The age factor plays an important role in the wound healing process. The most important factor in the wound healing process is the protein turnover rate. As the age increases this capacity of the body to turnover proteins is reduced considerably leading to slower wound healing rates.

Rests

Granulation tissue has a delicate blood supply that is easily damaged by movement and shearing forces. With early mobilization being desirable after surgery, the surgeon must consider carefully his advice to the individual patient to avoid both the hazards of prolonged rest and the discomfort of premature mobilization.

Infection

Once infection is established the fibroblasts must compete with the bacteria and the inflammatory cells for oxygen and nutrients. Thus, overall collagen synthesis is inevitably reduced and the collagen breakdown is enhanced by collagenolytic enzyme activity. Infections are a major factor in wound healing failure.

Malnutrition

Defective synthesis of both collagen and ground substance can be directly linked with malnutrition. Severe protein calorie malnutrition has long been implicated in the failure of wounds to heal, while lesser degrees of malnutrition depress healing as well. Vitamin C is necessary for the synthesis of ground substance. Vitamin D is essential for new bone formation and vitamin A for epithelialization. Calcium, zinc, copper and manganese are essential minerals and patients with burns and intestinal fistulas, in particular, become zinc depleted and require supplementation.

Uremia

Experimentally the addition of urea to tissue cultures of fibroblasts inhibits their growth. Clinically uremia is implicated in the retardation of wound healing in patients.

Jaundice

Jaundice is associated with reduction in wound strength. The appearance of fibroblasts and the formation of new blood vessels are both delayed. Biopsies of the skin in jaundice patients show a reduction of the enzyme proline – hydroxylase involved in the collagen maturation.

Steroids

An inflammatory response is essential for wound healing to proceed normally. Steroids depress wound healing by their anti-inflammatory action. New vessels are abnormal and sparse, as are fibroblasts. If steroids are given after the inflammatory phase of wound healing there is little overall effect of healing.

Radiation

Radiation causes cell death by damaging both DNA and disrupting intracellular metabolism.

AETIOLOGY AND PATHOLOGY

About 95% of the leg ulcers are due to vascular etiology (Gilliland) [13] and venous ulcers account for up to 90% of cases (Burton. S. Claude) [14], (Callam MJ, et al) [15] Arterial disease accounts for 5-10%; most of the others are due to neuropathy, usually diabetic or a combination of these diseases. (Yound JR) [16] Diabetic ulcers are common on the toe and the heel. (Hansson Carita) [17]

In European countries 90% of leg ulceration are due to venous disorders Occurrences of leg ulcers of various types are on the increase in par with the increase in longevity of life. 90% of the patients were aged 60 and above. In patients suffering from arteriosclerosis and hypertension, arterial ulcers are on the increase, especially in the western countries.

Arterial insufficiency and / or diabetes may also be the causatives for ulcers below the line of the shoe. Ulcers at the ankles in the gaiter zone and venous ulcers are mostly caused by venous insufficiency. (Hansson Carita) [17] Another important causative of venous ulcers is sapheno-femoral incompetence (94.4%). (Sethia. K and SG Drake) [18] Primary varicosity of the long saphenous system and / or short saphenous system is one of the causative factors for venous ulcerations. (Hoare MC et al) [19]

The elevated ambulatory pressure in peripheral venous system in chronic venous insufficiency manifests itself not only in form of disturbed microcirculation but also and particularly in microangiopathic changes. These include decrease in capillaries, glomerulus like changes and decrease in oxygen content. (Junger, M Stiens. A) [20]

It has been also noted that perivascular fibrin cuffs and skin hypoxia precede lipodermatosclerosis in limbs at increased risk of developing a venous ulcer. (Stacey M. C., Burnand K.G et al) [21]

Destruction of the dermis and the epidermis caused due to a lesion is termed as an ulcer. An ulcer can also be described as a breach of the skin or the mucous membrane with varying degree of infection. Venous and arterial diseases are the main causes of leg and foot ulcer. The cause of distribution may be a focus of inflammation and necrosis a short distance below the surface leading to disintegration of overlying skin and mucous membrane or, it may be in the nature of a shallow would which subsequently becomes infected.

Classification of leg ulcers

1. Venous ulcerations
2. Arterial insufficiency – Thromboangiitis Obliterans, atherosclerosis, Raynaud's disease and phenomenon, hypertensive ischemic ulcer (Martorell's ulcer) and embolic occlusions (sub-acute bacterial endocarditis).
3. Diabetic neuropathic ulcerations.
4. Neoplastic ulcers – epithelioma melanoma, basal cell carcinoma and malignant change in long standing scars (Marjolin's ulcer).
5. Tropical ulcers including leishmaniasis, fungal infections.
6. Specific infections – tuberculosis, syphilis and AIDS.
7. Blood dyscrasias – severe anemia, sickle cell anemia, thalassemia, hereditary spherocytosis and leukemia.
8. Nutritional and metabolic disturbances.
9. Skin sensitivity or allergy.
10. Trauma.
11. Necrosis by injection of chemicals, insect bites, snakebites or radiation.
12. Repeated trauma.
13. Rheumatoid arthritis.
14. Systemic autoimmune and micro vascular diseases. [22]

There are various stages in the evolution of the ulcers.

They are:

- The extension stage: Floor of the wound is covered in slough and the base is indurated.
- The transition stage: separation of the slough combined with the clearing of the floor of the wound and appearance of red granulation tissue marks this stage.
- The healing stage: transition of the granulation tissue into fibrous tissue is observed, which later contracts and the wound healing by epithelization is seen particularly in this stage.

The Venous Ulcer

Histopathologic reasons caused by venous insufficiency, is one of the main causes of venous ulceration. Changes taking place in the cluster of thick-walled capillaries within a thickened fibrotic papillary dermis, accompanied by siderophages and extravasated erythrocytes in variable numbers also contribute to the overall situation of ulceration.

Occurrence of perivascular fibrin cuffs in the skin is seen in venous insufficient areas. They are often present in the walls of superficial vessels and evident as smudgy eosinophilic areas. In venous ulcers it should be noted that the fibrin and the neutrophils are present in the walls of the vessels in the granulation tissue beneath the ulcers.

Lipodermatosclerosis or sclerosing panniculitis demonstrates fibrosis and thickening of the subcutaneous septa. An early lymphocytic infiltrate of the subcutaneous septa is gradually replaced by a mixed infiltrate with increasing fibroplasia and sclerosis.

In the late Lipodermatosclerosis there is minimal inflammation and subcutaneous sclerosis. For the normal movements to take place in the calf muscles of the leg several intricate and precise muscular movements need not take place. Dysfunction of the calf muscle pump can result in venous insufficiency in the deep, connecting or superficial veins; arteriovenous fistulae, or muscle dysfunction as a result of fibrosis, neuropathy or inflammatory diseases.

Valvular damage caused due to thrombosis may lead to deep venous insufficiency. Thus, dysfunctions of the calf muscle pump results in the failure to lower the venous pressure upon exercise causing venous hypertension.

Venous ulcers are mainly attributed to be caused by venous insufficiency; however multisystem incompetence of the valves is also a common cause. Increased venous pressure causes leakage of fibrinogen through widened endothelial pores. Due to this peri capillarial deposition of the fibrin occurs which in turn impends diffusion of oxygen and nutrients to the skin, causing ulceration.

Another plausible hypothesis suggests that reduction in the blood flow rate and endothelial damage allows white cells to form plugs, which in turn plug the capillaries. These cells release inflammatory mediators increasing vascular permeability. This causes tissue ischemia and ulceration.

Figure 7: Venous ulcer

Figure 8: Venous ulcer

Arterial Ulcers

Arterial or ischemic ulcers are most commonly due to atherosclerosis and hence encountered in older adults. They can be also seen in younger adults and here usually peripheral arterial disease like Thromboangiitis Obliterans is the cause. The other rare causes of ischemic ulcers are Raynaud's diseases and phenomenon.

Atherosclerosis develops at twice the frequency in patients who smoke compared with non-smokers. (Coffman J. D.) [23] about 50% of the patients have lipoproteinemia. Patients with diabetes develop the disease at an earlier age than non-diabetics and have more severe and progressive disease. The distribution of disease is also different in that diabetics have less aorto-iliac disease and more disease of vessels between knees and ankles.

There is focal accumulation of lipids, mucopolysaccharides, blood and blood products, fibrous tissue and calcium deposits in the intima of the arteries. Localized areas of thickening of the intima by fibroblastic proliferations and phagocytes laden with lipid materials are seen. The media becomes atrophic with thin strands of muscles, disrupted elastic lamella, collagen tissue and calcium deposits. Enlarging plaques encroach upon the lumen despite dilatation of the artery, and plaques may ulcerate. Hemorrhages occur within the arterial wall. Thrombi are formed and finally occlude the vessel lumen.

Another important feature here which causes sudden gangrene with ulceration is atheromatous embolization. It is due to embolization of small pieces of atheromatous plaques and debris to the arteries of extremities. This is called as blue toe or trash foot syndrome. Another important arterial disease, which can cause ulceration and gangrene, is Thromboangiitis Obliterans (Buerger's disease).

The etiology of TAO remains unknown. Almost all patients who develop this disease are smokers and the syndrome sometimes abates following cessation of smoking. An increased frequency of HLA-A9 and HLA-B8/B5 have been reported but not found by all investigators. (Millis J. L. et al) [24] The pathogenesis involves production of ischemia and all its manifestations by an inflammatory action of medium and small arteries of extremities and also obstruction by thrombi.

In acute stage there is pan vasculitis of the arteries and veins; the diagnostic finding being arterial thrombi with foci of micro abscess and giant cells. In chronic stage only fibrotic obliteration of arteries is seen and diagnosis can only be surmised since atherosclerosis is absent.

Figure 9: Arterial ulcer (TAO)

Figure 10: Arterial ulcer (Atherosclerotic)

Diabetic Ulcers

The various factors, which are the contributions for the cause of ulcers, are Hyperglycemia, microangiopathy, Neuropathy, Liability to infection and Alteration in blood flow. The precipitating factors are injuries and infection. Injuries could be physical injuries like penetrating wounds, disruption of skin and injury due to continuous localized blunt pressure. Thermal injuries are as a result of extremes of cold or heat.

In a diabetic condition of either type 1 or type 2, hyperglycemia may lead to surgical complications. In this condition it affects the basement membrane of the capillaries and cell permeability; interfering with the transfer of oxygen and nutrients to the tissues.

Along with decreased supply of leukocytes into this area and also high concentration of glucose in the tissue fluids which help in the growth of pathogenic organisms, it impairs wound healing. The vascular changes that take place during diabetes are responsible for ulceration of the foot. The principle finding in the vascular flow mechanism in diabetes is a marked decrease in blood flow as shown by Doppler waveform.

There is associated loss of the important diastolic back flow phase. These changes are also reflected in the high ankle / brachial Doppler ratio found in diabetes. The major cause of this blood flow change is the distal AV shunting and sympathetic dysfunction. The other cause being atherosclerotic stiffening of the vessels, sometimes this is associated with calcification of the vessel walls. The irregular pattern of blood flow definitely has an influence in the ultimate delivery of oxygen to the tissues.

The thickening of the basement membrane is characteristic of both variants of diabetes mellitus. There is either increased synthesis of basement membrane or decreased degeneration. Diabetic patients also tend to develop calcifications more commonly and at a much younger age. This causes some vascular narrowing which may cause distal ischemia.

Calcification of intima may produce trauma to blood cells and cause development of thrombi which may distally produce localized areas of ischemia and gangrene. The distribution of the atherosclerotic plaque and ulcers slightly differ from those is non-diabetics in that the disease primarily affects the large vessels of the lower limbs.

Figure 11: Diabetic ulcer

Figure 12: Diabetic Neuropathic Ulcer

Histologically the disease is manifested as a thickening of the tunica intima along with large atheromatous plaques and lumps. It is the involvement of the arteries in the foot which determine the healing of the minor wounds and extensibility of infections and necrosis. The metatarsal arteries are more often occluded in diabetes.

An important yet a very common factors responsible for producing neuropathic ulceration is the presence of diffuse distal peripheral polyneuropathy due to which painless ulcers are predominantly seen in the lower limbs. During this type of ulceration there is involvement of all types of nerve fibers. A notable feature is the marked reduction in the amount of Schwann cells. Neuropathy is predominantly selective for large myelinated fibers sub serving proprioception, touch and vibration senses.

Later small myelinated fibers and non-myelinated fibers sub serving pain and temperature sense are affected leading to mixed fiber neuropathy. Osmotic and metabolic derangements are caused due to ischemia from vessel occlusion and altered capillary permeability; this paves the way for producing neuropathy. There is no marked change in the arteries, which might account for the neuropathy.

Increased capillaries permeability is also another method which in turn leads to neuropathy. Increased capillary permeability allows the toxic proteins which are circulating to reach the nerves and tissue oedema which cause the impairment of nutrition contribution to development of the neuropathy.

Metabolic conditions prevalent are more important in the development of the neuropathy than are the vascular diseases. Abnormal Schwann cell function causes segmental demyelination, which is a dominant histological aspect of diabetic neuropathy. This hinders nerve conduction and the situation can only be reversed by remyelination. There are number of reasons which contribute to the production of myelin.

Some of these factors are:

Synthesis of abnormal myelin

Increased activity of polyol pathway in conversion of glucose to fructose with a resultant increase in concentration of metabolites. Simultaneous occurrence of axonal damage is also observed, but segmental demyelination of diabetic neuropathy may exist independent of axonal damage. Axonal damage is the first manifestation of diabetic neuropathy.

Normal nerves contain high concentration of myoinositol and a disturbance in its metabolism in diabetics is suggested as the mechanism for axonal damage. Reduction or absence of sensation increases the changes of injury occurring to the foot from heat, sharp objects, pressure from ill-fitting shoes etc. In the most severely affected patients, the chances of spreading infection, formation of large ulcers and gangrene is increased tremendously. The motor changes are primarily manifested in the small muscles of the foot. Metatarsophalangeal joints are hyperextended when the small muscles of the feet are paralyzed with preserved function of the long flexors and extensors. Because of this condition there is a predisposition to the development of ulcers, especially at the tip of the toes from the abnormal pressure. Due to autonomic neuropathy, there is a failure of vasodilatation producing hypoxic environment predisposing to infection in minor injuries. The absence of the sweat glands or its malfunction, with the loss of Lysozymes alters the cutaneous bacterial environment and also decreases the defense mechanism of the skin. Trophic changes include redness and glossy shining skin with loss of hair. Thick keratinization of the skin and atrophy of the local subcutaneous tissue is observed in areas of increased pressure. Breakdown of such skin produces trophic ulcers.

Tropic Ulcers

Tropic ulcers are particularly perforating ulcers of the foot, which is associated with Tabes dorsalis, diabetic neuropathy, leprosy, and other diseases of the central nervous system. About 10 to 20% of the patients with leprosy all over the world complain of the most common yet the most serious complication that is of ulceration on the bottom of their feet.

There are various types of ulcers that are seen in leprosy are the lepromatous skin nodules. The second type of ulcer which is seen here is the stasis ulcer which is seen in the crease of flexor muscles of the ankle. This is due to autonomic dysfunction leading to abnormality in regulation of blood supply and absence of sweating. The last and the third type of ulcer seen in leprosy is the plantar ulcer, which is seen in the weight bearing areas of the foot where there is sensory loss. (Dharmendra) [25]

The three modalities of neurological deficit are:

1. Sensory loss
2. Motor paralysis
3. Autonomic nerve damage
4. Sensory loss

Impending danger to the tissues is preceded by the sensation of pain, which is the body's natural alarm system. But in the decreased condition due to the neurological impairment (sensory loss), repeated trauma may attain proportions which might actually cause tissue destruction.

Motor Paralysis

Only open injuries on the foot with intrinsic paralysis are usually liable to be ulcerated, whereas the foot prone to ulcers always shows motor as well as sensory loss. The pads of the fore feet are protected by the intrinsic muscles of the foot in two ways, one by straightening the toes, the metatarsal heads are slightly retracted and the pulps of the terminal segments also take a small share of the weight. When these muscles are atrophied the bony prominences are made more obvious.

Autonomic nerve damage

Blood supply

The regulation of the blood supply to the sole of the foot is largely, reflex, depending on the integrity of sympathetic supply to the muscular part of the arteriole. In the nerve trunk lesions, these nerves are also destroyed so that the arterioles are sympathectomized. In an anesthetic foot, the increase in blood supply after exercise or slightly excessive stress is not present and hence there is a relative tissue anoxia ensues. Repeated episodes of such anoxia result in increase of fibrous tissue and a reduced tolerance to infections.

Sweat loss

The second autonomic function that is disrupted is the control of the sweat glands. Thus, the denervated foot is dry. Due to this deprivation in the supply of the sweat, another one of the skins protective mechanisms is lost, which causes the skin to lose its suppleness. Deprivation of sweat causes the skin to go dry and crack, which may cause the introduction of infections into the body. This kind of skin cracking can be viewed most commonly in the weight bearing areas where special mechanical strains are supplied during normal walking.

Studies have shown that the initiation of the ulceration is usually due to an initial damage in the deep tissue surface. The pressure of the entire body falls on the foot and during walking, unperceived by sensation and the normal rotation of the joints is uncontrolled by muscular coordination, the result being the introduction of torsional stress, which leads to necrosis of the deep tissues immediately adjacent to the bone.

Figure 13: Topical ulcer on Heel

Figure 14: Topical ulcer on Base of Great Toe

The clinical features follow a sequence of incidents which are listed below:

1. Initial deep necrosis.
2. Necrotic sinuses.
3. Necrotic blisters and finally.
4. Plantar ulcer.

DIAGNOSIS

Chronic leg ulcers can be diagnosed based on the patient's medical history, appearance and location of the ulcer along with clinical examination and investigations. History is very useful in diagnosing ulcers caused by associated diseases.

The diseases that have to be specially inquired for are Diabetes Mellitus, Syphilis, Tuberculosis, Nephritis, Liver disorder and Blood disease. The primary lesion might have been altered either by the use of ointments or by infections. Thus, a history of eczema helps for diagnosis of stasis ulcers. History of trauma will help for evidence of underlying fractures or osteomyelitis. A nodular ulcer appearing after a thorn prick points to deep mycosis. A painful ulcer with undermined edge or ulcer healing at one end and growing at other end might be of tubercular origin. Ulcers growing on already existing moles may be malignant melanoma. Rolled out borders of chronic ulcers may be carcinomatous ulcer. Ulcers growing on scar tissue may be Marjolin's ulcers.

Punched out ulcers without any symptoms in elderly people may be due to gumma. Knowledge of precipitating factors is also equally important. Ulcers that get aggravated during cold season indicate Raynaud's disease. Thus, a clear idea of primary lesion of an ulcer and its later development helps one in diagnosis. An understanding about the location of the leg ulcer is crucial. The area around the medical malleolus is drained by long saphenous venous system and communicating vein of lower limb and this is the area where occurrence of stasis ulcer is invariably seen. Hypertensive ulcers or ischemic ulcers are usually pale with an eschar and are usually seen on the lateral side.

Figure 15: Traumatic ulcer

Figure 16: Ulcero -Proliferative Growth

Hyperpigmentation and or eczematization point towards stasis element. In Werner's disease or scleroderma, sclerotic skin formation is observed. Distinguishing between a spreading ulcer and a healing ulcer is extremely important. Inflamed and edematous edges of the ulcers indicate spreading ulcers.

In a healing ulcer, the edge is traced from red granulation tissue and at the center it will show a blue and a white zone. An undermined edge is usually observed in a tuberculous ulcer and punched out edge in gummatous ulcer. A pearly white beaded edge which is raised is characteristic of a rodent ulcer. An everted edge points to squamous cell carcinoma. An understanding of the floor of the ulcer or exposed part of the ulcer is extremely important. Red granulation tissue covering the floor of the ulcer points to a healing ulcer. Smooth granulation with a pale coloration indicates to a slow healing ulcer. Trophic ulcer penetrates down to the bone which becomes the floor in this case. Black mass on the floor suggests malignant melanoma.

Figure 17: Healing ulcer with red granulation tissue

Discharge

The quantity and the coloration of the discharge from an ulcer help in identifying the nature of the ulcer. Profuse discharge from an ulcer indicates to a healing ulcer whereas a purulent discharge points to a spreading ulcer. A greenish coloration to the discharge indicates to an infected ulcer and this may be caused by pyogenic bacteria. Serosanguinous or blood mixed discharge is pathognomonic of malignant ulcer, or else tuberculous ulcer.

CLINICAL FEATURES AND DIFFERENTIAL DIAGNOSIS

Arterial ulcers are rare compared to the venous ulcers. An arterially compromised limb can be pale and cyanotic. The skin is often dry and scaly or shiny and atrophic with brittle nails or loss of hair growth. The appearance of the ischemic erythema is usually on the foot. Atherosclerotic arterial ulcers are often seen in older people and when they occur in the young adults, it usually points to Thromboangiitis Obliterans and other forms of vasculitis.

Pressurized areas of the foot are the focal points for the appearance of these ulcers. Toes, bony prominences or in pretibial and lateral malleolar areas are some of the common places where these ulcers can be spotted. These ulcers have sharp defined edges and are punched out. The ulcer destroys the deep fascia and may even expose the tendons at the base.

The ischemic ulcers are extremely painful and the pain is excruciating irrespective of the size of the ulcer. The development of the arterial ulcers is more prolonged when compared to the venous ulcers. The appearance of ulcers having punched out edges with slough deployment on the floor of the wound resembling a gummatous ulcer points directly to a trophic ulcer.

The most common forms of trophic ulcers seen are the bedsores. The heel and the ball of the foot and the back of the heel are the most common places where these types of ulcers occur. These ulcers start with callosity under which suppuration takes place, the pus comes out and the hole at the center forms the ulcer which gradually burrows through the muscles and the tendons to the bone. Tuberculous ulcer develops when cold abscess from bone and joint tuberculosis breaks out of the surface.

Appearance of the ulcer is in the form of a thin edged one wound, which is reddish blue in color and undermined. The base of the wound is colored pale and there is slight induration at the base. The areas where gummatous ulcer appear most commonly is in the upper part of the leg i.e., over the subcutaneous bones of the tibia.

The wound has punched out edges and a yellowish grey gummatous tissue (wash lather slough) on the floor. Erythrocyanoid ulcer is associated with 'erythrocyanosis frigida', which is found to occur exclusively in young aged women. Abnormally fat subcutaneous fat with thick ankles combined with abnormally poor arterial supply are the contributing factor for the formation of this type of ulcer. The skin of the ankle tends to become abnormally sensitive to temperature in such patients. When the temperature drops the ankle becomes cold and blue and often tenderizes.

In hot weather chronic reactive hyperemia is evident and the ankle becomes hot, edematous, swollen and painful. The patient is much more troubled by chilblains. Palpation of the leg reveals the presence of small, superficial and painful nodules which are numerous. These nodules break down to form ulcers. The hypertensive ulcers or the Martorell's ulcers are a condition which appear in older aged people and is usually associated with atherosclerosis. This ulcer is extremely painful and is usually punched out and extends down into the deep fascia. It is not worthy that all peripheral foot pulses are present. The clinical characteristic feature of the Meleney's ulcer is the burrowing nature, which is the undermining of ulcer with a lot of granulation tissue on the floor. These ulcers are painful and show a tendency to spread and render the patient toxemic. Ulcers secondary to Paget's disease are mostly situated over the convexity of the anteriorly bowed tibia, with the edges densely adherent to the bone which forms its base. In case of rheumatoid arthritis, the ulcer forms due to break down of the nodules.

The size of the ulcer varies, the wound becomes punched out, shallow and is usually painful, without induration and slow to heal. In case of hematogenic ulcers, it usually mimics venous ulcers or ischemic ulcers and are commonly found to occur in anterior part of the leg. In patients suffering from non-specific yet chronic ulcers, the patients usually have a history of trauma which he might have neglected or for which he had taken inadequate treatment.

The patients in such cases are of poor build, poorly nourished and anemic on examination. These ulcers appear with irregular margins surrounded by oedema and there may be slight tenderness. Often ulcers are foul smelling and with seropurulent discharge. Associated induration, sclerosis and pigmentation are common. Yet another type of ulcers usually occurring in older aged, bed-ridden patients is the Decubitus ulcers. Young people with neurologic disease can also be affected with this type of ulcer. The ulcer localizes over bony prominences, the heel region is one of the characteristic sites. These ulcers are present as deep ulcers and are marked with peripheral undermining which can extend to the bone, causing osteomyelitis.

MANAGEMENT

INVESTIGATIONS

There have been many investigations done for analysis and understanding of the leg ulcers. These investigations are numerous and varied. These investigations help us to come to a definite diagnosis of the ulcers and may also guide about the outcome of surgery if any.

The investigations can be broadly classified in to three different classes:

1. Routine investigation.
2. Special investigation.
3. Investigation to know vasculature status of the leg ulceration.
4. Routine investigation

The routine investigations themselves are comprised of the following tests:

Hemoglobin (Hb%)

This test helps to determine whether the patient is anemic. This is extremely important in the wake of the information that the leg does not receive enough blood supply when compared to other parts of the body.

ESR

ESR is increased in many conditions of various types, but ESR more than 30mm/hr. makes one to think about chronic granulomatous disease. The ESR content in the body is raised in tuberculosis, sarcoidosis deep mycosis, SLE, dermatomyositis etc. But ESR has only prognostic value.

TC and DC

Increased total count always denotes the increased activity of the reticuloendothelial system. It represents acute or chronic infections. In some cases, it can be of vital importance as in Leukemia. But by itself TC is not sufficient for diagnosis. DC is also taken along with TC, Differential count talks about the type of cells in increase and also the presence of abnormal and malignant cells. Increase in percentage of lymphocytes indicates chronic infections or allergic phenomenon.

Hemogram

Any abnormal findings from the above investigations may call for a complete hemogram to know the further details of the blood cells, e.g., Sickle cell anemia.

Special investigation

Many different types of special techniques are also used which are classified in the special investigation group.

Fasting blood sugar, PPBS, and GTT

These tests are done to determine the presence of diabetes mellitus. As we know diabetes mellitus is one of the many contributing factors for the formation of ulcers in legs and for peripheral neuropathy. The ulceration process is hastened in the presence of higher sugar concentration in the blood.

Bone Marrow

Bone marrow aspiration is recommended in cases where leukemia or aplastic anemia is suspected. The use of the sternal bone for this test is usual practice. Presence of abnormal blood cells and an increase in the WBC content and their precursors help in the process of diagnosis.

Lipid profile

The detection of the atherosclerotic condition is possible when this particular test is performed. As we know atherosclerosis by itself can be a causative agent of ulcers. It has been noted that people without arterial disease maintain an LDL cholesterol level of 130 mg/dl or less. An LDL level of more than 160mg/dL must be treated with lipid lowering drugs.

Lupus Erythematous cell test

The presence of systemic lupus erythematosus is confirmed by performing this particular test. In recent times this test has been replaced by other tests such as anti-DNA antibodies test and L.E band test. The test is very helpful in detection of lupus Erythematosus and other tissue disorder.

Blood VDRL test

The VDRL test is a non-specific flocculation test used for diagnosis of syphilis, Yaws Pinta and Bejel. The test depends on the presence of Regain in blood and it cannot differentiate Yaws or Pinta from syphilis.

Liver function test

The status of the liver and its functioning is known by the performance of this test.

Culture and sensitivity of Bacteria/fungus

The pus from the ulcer is sent to the lab for culture and sensitivity for bacteria or fungus as the case may be. The presence of these pathogens in the pus does not have to mean that they are the causations. These pathogens only act as catalysts and worsen the condition and hinder essential

processes such as coagulation and fibrinolytic activity from taking place. Presence of other bacteria must be taken more seriously.

These bacteria are:

1. Hemolytic Streptococci.
2. Fusiform bacilli and spirochete in hot humid conditions.
3. Candida albicans.
4. Pseudomonas aeruginosa.
5. Proteus
6. Staphylococcus

Presence of these organisms needs attention. They have to be treated with proper antibiotics.

Biopsy and histopathological examination from ulcer site

Malignant skin changes are common in chronic leg ulcers. A biopsy should be taken from all suspicious ulcers or ulcers that do not respond to appropriate treatment. Also, chronic ulcers need to be biopsied at regular intervals as malignant change in these ulcers in directly related to their duration. (Yang D. Morrison B. D. et al and Smith J, Mello I. F et al) [26],[27]

Radiological examination

X-ray should be taken to find out any bony abnormalities and or osteomyelitis of the underlying bone.

Investigation of vascular structure of the leg

Investigation of venous disease

Doppler ultrasound

A Doppler flow probe can be used to exclude arterial disease and to determine patency of a vein and a bi-directional probe is used to detect any reflux. This investigation is carried out with patient standing. Doppler probe is first placed on saphenofemoral junction (SFJ) and blood flow is assessed to located venous flow in common femoral veins.

With one hand the examiner gently heard as “woos” from loud speaker of the Doppler machine. The calf compression is released and any backflow is noted. The probe may be also held in sapheno-popliteal junction (SPJ) while calf is compressed and released to test competence of veins in this region.

Duplex ultrasound imaging

This technique involves use of high-resolution B-mode ultrasound imaging and Doppler ultrasound to obtain images of veins and simultaneously measure the flow. It provides both functional and anatomical information. Modern duplex machines represent flow as a color map which is superimposed on grey scale image of the vessel. This technique is highly reliable in the investigation of arteries and veins, and is most appropriate when detailed analysis of anatomy and physiology of venous system is required. Blocked or incompetent veins can be easily identified. The origin of varicose veins and venous ulceration can be identified and in patients with deep vein thrombosis the thrombus can be seen. (Seurr John. H et al) [28]

Venography

This investigation is X-ray equivalent of Duplex ultrasonography. It has been largely replaced by duplex imaging.

Plethysmography

Photo- Plethysmography: in this investigation a probe is attached to the skin to asses venous refilling of surface venules by measuring light transmission of the skin. The filling of vessels reflects pressure in the superficial veins of the leg. The patient sits till the trace stabilizes. Then he performs a series of 10 dorsiflexion at the ankle. The venous pressure falls as venules empty and the trace falls. The patient sits and venous refilling occurs only through arterial inflow, a slow process taking 20 to 30 seconds when the limb is at rest.

In venous incompetence filling occurs through venous reflux also which speeds the filling time. This test can be repeated after applying tourniquet at the thigh to occlude long saphenous system and below the knee to occlude short saphenous system to know which set is incompetent. Other forms of Plethysmography like air plethysmography, light reflex rheography and strain gauge plethysmography are used only by experts in laboratories to quantify venous function caused by incompetent veins. (Seur John. H et al) [28]

Investigations for arterial disease Doppler ultrasound technique

The frequency of the ultrasound waves is altered when it bounces off moving objects and the change in the frequency can be directly related to the velocity with which the object is moving, this effect is termed as the Doppler's effect. RBCs in the blood act as perfect reflectors. The change in the frequency of the ultrasound is recorded into audible sound or on paper. This technique is extremely accurate and can easily pin point femoral and popliteal or iliac obstruction.

Duplex imaging (Duplex ultrasound scanning)

This is an investigative technique of major importance in vascular disease. A duplex scanner uses 'B' mode ultrasound to provide an image of vessels. This image is created through the different ability of different tissues to reflect the ultrasound beam. A second type of ultrasound, namely Doppler ultrasound is then used to isolate the imaged vessels and the Doppler shift is analyzed by a computer in the duplex scanner itself the various colors indicate change in direction and velocity of flow; points of high flow generally indicate a stenosis. It also allows the cross-sectional area of arterial lumen to be measured. By use of color, the flow towards or away from the transducer can be easily distinguished so that peripherally running arterial flow (red) can be immediately distinguished from centrally directed venous flow (blue); the intensity of color increases with the velocity of flow. Reversal flow, in venous reflux can be recognizable by a change in color, so that incompetent veins and leading walls show up as 'red' at the moment of reflux. Duplex scanning is feasible and reliable in detecting crural and pedal artery lesions in lower limb with severe ischemia. (Karacagil. S. Lofberg. A. M) [29]

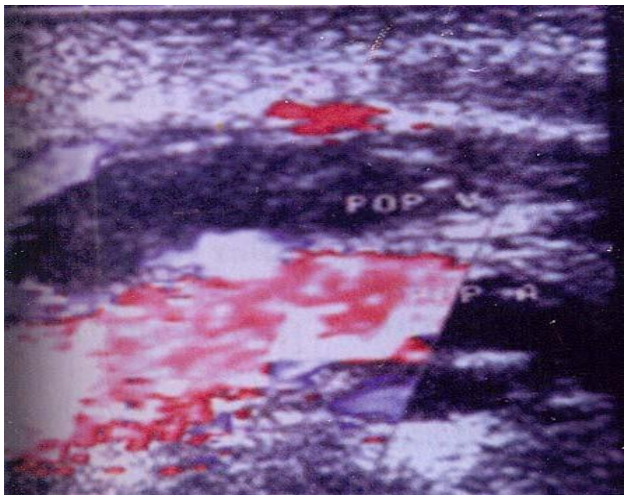


Figure 18: Thrombus in popliteal vein

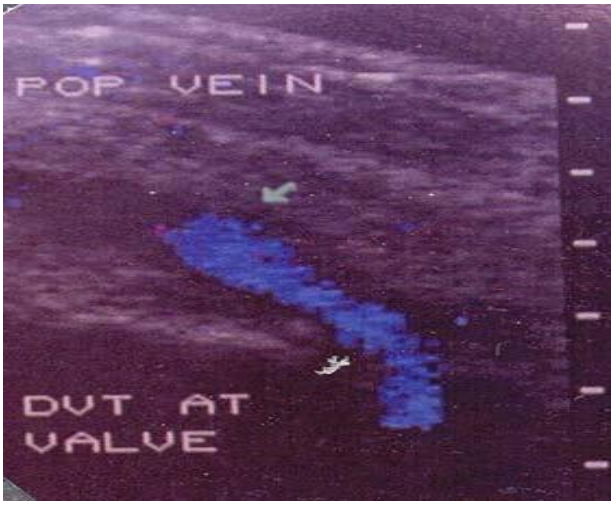


Figure 19: Popliteal vein with thrombus near the valve

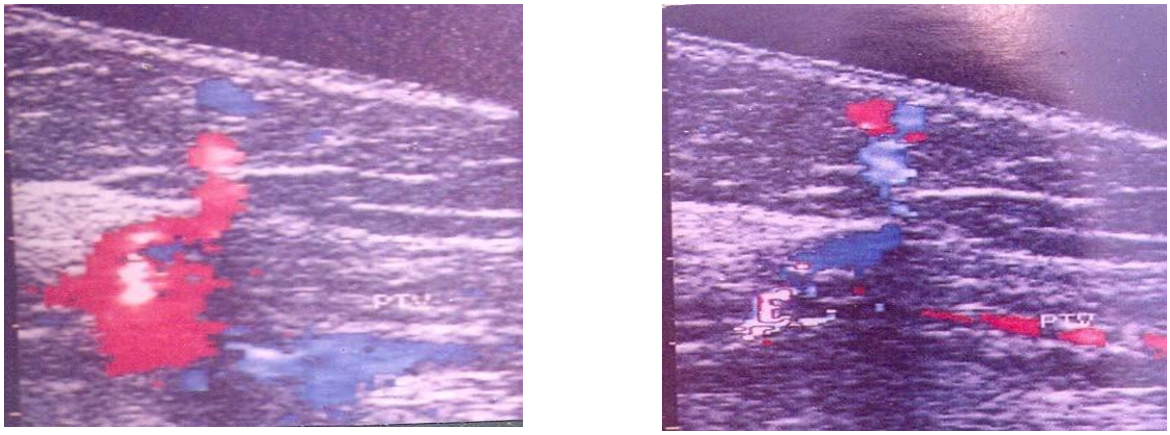


Figure 20: With distal compression of foot, flow is normal from superficial to deep vein (coded red), on release flow is retrograde through the perforator (coded blue)

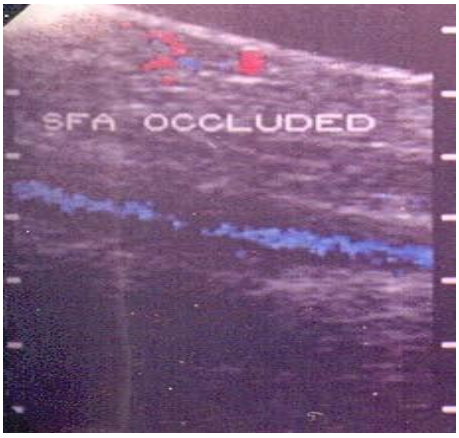


Figure 21: Superficial artery occlusion

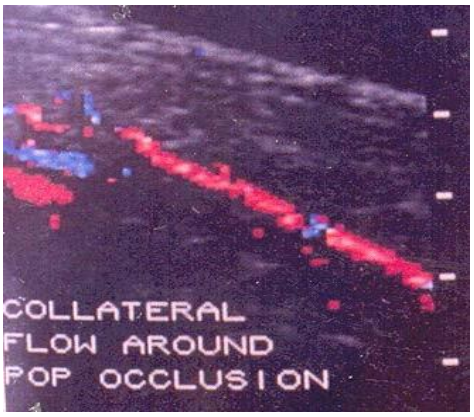


Figure 22: Popliteal artery with collateral flow around

Radioisotope tests

Fibrinogen labelled with radioactive isotope I125 is injected into the patient. This is converted into fibrin by thrombin. Fibrin is deposited in thrombus. Various other tests are also performed in this type of special investigations. Some of these are mentioned below:

Arteriography

Arteriography is indicated only when decision has been taken that intervention is needed. Arteriography involves injection of radio opaque solution into the arterial tree using retrograde percutaneous method involving femoral (Seldinger technique). Direct angiography by puncturing the aorta is hazardous and outdated. Hazards include the thrombosis, arterial dissection, hematoma and anaphylaxis. (Murie. John) [30]

Digital Subtraction Angiography (DSA)

This technique involves a computer system to digitize angiographic information. It allows image before contrast injection to be subtracted from contrast image, yielding great clarity. This avoids arterial puncture although high volumes of contrast agents are required.

Plethysmography

This assesses volume change in a limb with each cardiac cycle. Air filled cuffs or mercury in rubber strain gauges have been typically used. For most clinical purposes the test has been superseded by duplex imaging. (Murie John) [30]

TREATMENT

The main aim of treating any ulcer is to make it heal and to treat its underlying conditions. Various methods of treatment are prevalent today. The treatment given to the patient can be classified into three general classes. They are explained in detail below. General Treatment In treating the ulcer, the surgeon must first recognize and treat any systemic condition that inhibits the healing. Systemic diseases, particularly malignant tumors, diabetes mellitus, nutritional deficiencies and some medication can retard the wound healing. In addition, deficiencies of vitamin C, zinc, iron and protein can also retard healing as these are the nutrients needed to synthesize collagen. It is important to keep in mind that some medications like systemic glucocorticoids; cytotoxic drugs and non- steroidal anti-inflammatory drugs if administered may cause impairment to the process of collagen production which intern hampers the wound healing process. The general practice of treatment of the patient in this condition must include rising of the patient's limb to an elevated position and performance of exercises within bandage supports. The elevation of the affected limb well above the heart level is an excellent treatment method and the limb must be allowed to remain in this state for at least 2 hours. The surgeon must encourage the performance of exercise. The exercises need not be too vigorous and might just be simple ones as rotation of the ankle through full range and gentle lift of the leg off the floor. The rate of exercise should be around 40/min and this is enough to empty the veins without overloading the lymphatic system. As a rule, elevation must not be done in cases of ulcers with arterial origin.

Systemic treatment

Infection of the ulcerous wound greatly hampers the wound healing process and keen attention must be paid when dealing with such cases. The treatment of such ulcers must employ the use of proper medication in the form of antibiotics. Oral agents such as decloxacillin are appropriate treatment for gram positive infections, while Amoxycillin, clavulanate (Augmentin) will extend bacterial coverage to some gram-negative organisms and anaerobes but not to pseudomonas, which can be effectively treated with Ciprofloxacin.

Oral Zinc Sulphate Therapy

The value of this method in the process of ulcer treatment remains debatable. But it is reasonable to treat patients who have shown to have low plasma levels or where there is a background of malnutrition or an inadequately balanced diet, prolonged anemia or toxemia. A dose of 220 mg twice a day is well tolerated.

Vasoactive medication

Some vasoactive agents have been found to be effective in various conditions. Nifedipine is effective in Raynaud's disease as a result of its ability to restore blood flow to the digits. Ketan serine, an S-serotoninergic blocker is helpful in treating ischemic ulcers. Pentoxifylline (trental) is effective in treatment of the above conditions. Other physical modalities of treatments such as transcutaneous electrical nerve simulation may also act by increasing blood flow. Ketan serine has also been shown to increase the rate of ulcer healing in diabetic ulcers. (O'Meara S.O. Cullum N et al) [31]

Local treatment

Local application

This method is practiced to soak up exudates, remove slough, control infection, sooth the surrounding skin, promote granulation tissue and protect seeding of new epithelium as well as to relieve pain. The effect of this type of treatment remains marginal or insignificant if other methods such as elevation, exercises are not administered.

It is not always possible to heal an ulcer completely, so the next most logical thing must be done that is it should not be allowed to get worse. To accomplish this relatively bland agents such are used on the ulcers to control the incurrence of infection. The use of topical antibiotics must be restricted as most of these are strong sensitizers. The use of antiseptic agents is safer and acceptable.

Wet dressing using Eusol or 0.5% Acetic acid, chlorhexidine or 0.5% silver nitrate have been shown to be inhibitory to granulation tissue, and must be advised with caution. However, Eusol is a useful wetting agent for softening and removing superficial slough, especially when there is no granulation tissue. 0.5% silver nitrate is recommended when the granulation tissue is over exuberant.

Cautious irrigation with saline or repeated application for the removal of saline soaked gauge may be helpful. Gilliland E. L and Wolfe John H.N. [13] recommended povidone as the antiseptic for general use and silver sulphadiazine for short periods of pseudomonas species have been cultured and potassium permanganate (1/8000 dilution) for wet ulcers with surrounding eczema but again only for limited periods as it can cause local hyperkeratosis.

In recent time's new topical dressing have gained wide acceptance in the treatment of leg ulcers. These products can optimize wound healing by providing moist environment, which aids keratinocyte migration over the surface of a wound. The hydrocolloid dressings are to debride the ulcer, while some of the polyethylene dressings have the advantage that they decrease the pain in the ulcers. These dressings do not increase the risk of infection in the ulcer colonized with bacteria. Carboxy-methyl-cellulose dressings are safe, effective and well tolerated in management of non-ischemic, non-infected deep diabetic foot ulcers. (Piaggesi A., Baccetti F et al) [32]

Compression bandages

Bandages are either used to compress or to retain dressings, support and contain any prior reduction and swelling. The advantage of compression lies in the provision of adequate counter pressure when the patient continues normal occupation and normal life. Full use of natural leg pump is encouraged.

Several methods of compression have been used. It probably maters less which method is chosen that it is carried out with expertise and that its limitations are known. Recently the group from Charing Cross Hospital has achieved 80% of ulcer healing within 6 weeks using a high compression 4-layer bandage. Generous soft padding is necessary to distribute evenly the high pressure so acquired.

Compression is more effective in healing venous leg ulcer than is no compression and multilayered high compression is more effective than single layer compression. High compression hosiery is more effective than moderate compression in preventing ulcer recurrences. (Callam N, Nelson E. A et al)

SURGICAL TREATMENT

Many ulcers that are infected and do not heal require wound toileting debridement and slough excision. Those ulcers which still do not heal by secondary intension or have delayed healing or those which have large areas of defects require skin grafting.

Grafts

Primary indication for surgical treatment is the failure of conservative line of management. It is also helpful in cases of extensive ulcers which when treated conservatively will take long drawn-out period for healing; that too with fibrosis and scarring.

Pinch grafting

It is relatively a painless procedure that consists of removing small "pinches" of skin, partial thickness grafts, under local anesthesia from a donor site, e.g.: the thigh. These are then applied dermal side down to the wound bed. The procedure is indicated in patients with medical problems that might preclude general anesthesia. The recipient site usually heals with a "cobble stone" appearance and the technique is not practical for large ulcers.

Split thickness grafts

These can be used in larger ulcers. It is advisable to use a graft that allows the underlying build-up of exudates to escape. This technique usually requires extensive local or spinal anesthesia. The donor site may be painful and slow to heal. The meshed graft heals by secondary intension, which results in irregular surface of the grafted site.

Keratinocyte grafts

Cultured epithelial grafts were first shown invitro by Ronwall and Green and first used clinically by Gallico in 1984. Here only epithelial component can be cultured and dermal component is lacking. Their lack of dermal component restricts adherence and attachment of underlying tissue reducing long-term durability of the graft. Work is also being done on dermal substitutes that can be used as temporary dressing. A combination of epidermal, dermal skin substitute holds great promise, at present, for future success. (Henry C. Vasconez) [34]

Autografts

Human keratinocytes from a small skin biopsy specimen can be grown in vitro, in 2 to 3 weeks, to form sheets of confluent epithelium. Cultured autografts have also been applied to chronic non-healing ulcers. They seem to provide rapid healing in some cases.

Allografts

Culture of epithelium derived from an allogenic donor (new born fore-skin) and subsequent application to chronic non-healing ulcer are fairly recent developments. Cultured allografts have been found to promote healing in burns. The new born keratinocytes appear to provide a potent stimulus to heal difficult skin ulcers of varying etiology. Cryo-presentation of these allografts allow storage of readily available grafts in skin banks. Cultured allografts may prove to be useful alternative for this treatment of chronic ulcers. In chronic wound such as ulcers, however both auto grafts and allografts seem to act as temporary biologically active wound dressings.

It is likely that growth factors released by the keratinocytes stimulate healing Also cultured epidermal allografts hasten healing of refractory leg ulcers apparently by stimulating the migration and / or proliferation of acceptors keratinocytes rather than through take up of the graft. (Beele H., Naeyaert J. M. et al) [35]

SPECIFIC TREATMENT

Venous ulcers

Specific treatment of venous stasis diseases must first be directed towards reducing venous hypertension. Patients with venous stasis diseases should wear support hose, but for patients who cannot wear support hose, treatment with Unna boot has shown to help healing venous stasis ulcers. These boots consist of zinc oxide, calamine and glycerin. The hypertension can also be reduced through the use of intermittent pneumatic compression.

For stasis ulcers that are refractory to these treatments, some of the surgical approaches include excision of the incompetent valves, leg valves reconstruction or transplantation of the brachial vein valve to leg veins. Once the ulcer has healed, to prevent recurrences specific surgical procedures in form of saphenofemoral ligation (Trendelenburg's operation) subfascial ligation, incompetent valve reconstruction will become necessary. A bypass operation may be performed for thrombosis.

Arterial ulcers

The therapy must be individualized. If the ulcers result from the localized occlusion of small arterial branches, then some will heal solely with bed rest with elevation of the head end of the bed and cool saline compression. Others will respond to lumbar sympathectomy combined with complete excision of the ulcers and its surrounding rim of infracted tissue followed by a split thickness skin graft. Occasionally if arterial bypass surgery may be warranted, it is of utmost important to determine the arterial inflow at the iliac level is adequate before attempting reconstructive arterial surgery on the femoropopliteal system.

Transluminal angioplasty may be performed for occlusive disease by inserting a balloon catheter into an artery and inflating it within a narrowed area. Balloon angioplasty has had its major success in dilating the iliac vessels. To a lesser extent the vessels of the leg itself may be dilated with a good outcome followed by stents. Atherectomy can be performed by a variety of new devices available to allow the percutaneous removal of atheroma from within the vessel.

Diabetic ulcers

Proper assessment of the diabetic status, severity of infection and general nourishment is the key to the success of management of ulcers. Regardless of the type of diabetes mellitus, they are suffering from (IDDM or NIDDM) all patients must be put on soluble insulin therapy. Depending upon the blood and urine sugar measurement doses should be adjusted. Control of infection is achieved by using combination of broad-spectrum antibiotic agents.

Pre malignant ulcers

Wide excision is the treatment of choice once the diagnosis is confirmed by biopsy. Excision of the growth should be performed with 2 cms of the normal tissue surrounding the premalignant growth in width and depth. The defect or the gap of wide excision is covered either by split skin graft taking skin from the contralateral limb, as skin grafting from the ipsilateral side may result in recurrence or with myo-cutaneous flaps and microvascular free tissue transfers.

Treatment

Initial line of treatment was to improve the general physical conditions of the patients. Anemic patients were put on iron preparations and some patients needed blood transfusion. If patients were dehydrated, fluid and electrolyte balance were restored by appropriate fluids like ringer lactate and isolyte solutions and vitamins were infused along with the fluids. Intravenous protein preparations (Amino drip), and intralipid infusions were instituted if required.

TAO patients were advised to quit smoking and were put on peripheral vasodilators like pentoxyphyllin and most of the patients had undergone indirect arterial surgery (Lumbar sympathectomy). Some patients had undergone above knee or below knee amputation. Patients who were suffering from varicose ulcers were first treated for ulcer with limb elevation, compression bandaging (Bisgaard's line of treatment). Later the patients were posted for surgery - Trendelenburg's operation (i.e., flush ligation of S.F. junction) and stripping (Meyers stripping); if there is saphenofemoral incompetence or multiple ligation and excision of vein, if perforators are incompetent. Some of the patients who were not ready for surgery were treated conservatively with further compression bandaging. (Elastocrepe bandage).

Diabetic patients with ulcers were put on simple diabetic diet which was rich in proteins and vitamins. Urine sugar was kept as near the base line as possible with insulin therapy. Plain insulin (Bovine) was initially used according to sliding scale and later a fixed dose was maintained in consensus with the physician. Local treatment of lesion by cleaning and dressing was done.

Slough excision was done, pus and discharge were sent for culture and sensitivity, and treated with antibiotics accordingly. Various local ointments used in the treatment included that of povidone-iodine, collagenase ointment (salutyl) and antibiotic containing ointments (Metrogyl ointment). Neuropathy was treated with neurotropic drugs.

Patients with atherosclerotic occlusive disease and increased lipid profile parameters were treated with lipid lowering drugs in consensus with the physician. They were further referred to vascular surgeon for direct arterial interventions.

Amputation was resorted to in cases of extensive gross infection and tissue necrosis, where it had been done as a lifesaving procedure. Pre malignant ulcers were confirmed with edge biopsy and were treated with wide excision. Raw areas were covered with split skin graft.

Some of the traumatic ulcers were also treated with split skin graft after controlling infection with suitable antibiotics. By the time of discharge some cases with residual wounds were advised to come for dressing twice a week. Diabetic patients were suggested to attend diabetic clinic regularly after discharge from the hospital.

MATERIALS AND METHODS

MATERIAL AND METHODS

Study Area- Department of General Surgery, Santosh Medical College & Hospital Ghaziabad

Study Population- All the patients with lower limb ulcers attend surgery OPD and admitted in surgery ward.

Study Period – July 2021-June 2022.

Sample Size- 65 Patients.

Study Design- Prospective interventional study.

Study technique- Convenient technique.

Inclusion Criteria: -

1. Patient willing to participate in the study.
2. Patient suffering from any type of lower limb ulcers.
3. Patient of age group 18-65 years.

Exclusion Criteria: -

1. Patients' refusal to be a part of study.
2. Patients <18 years and >65 years of age.
3. Immunocompromised patients.
4. Patients with suspected malignancy.

In this study, we are conducting a clinicopathological examination of ulcers on the lower limb and we are also discussing about its management.

In this context past researches provide evidence about the estimated prevalence proportion of leg ulcers from 0.18% to 1%. Hence, we have assumed the higher limit of this range i.e., 1% as the prevalence of leg ulcers for sample size calculation. On calculating sample size at 99.99% confidence level with 5% margin of error and prevalence 1%, it comes out equals to 65 patients. Hence, as we have described above, we have to choose 65 patients suffering with leg ulceration to proceed with this study.

Population size (for finite population correction factor or fpc) (*N*):	1000000
Hypothesized % frequency of outcome factor in the population (*p*):	1%+/-5
Confidence limits as % of 100(absolute +/- %) (*d*):	5%
Design effect (for cluster surveys-*DEFF*):	1

Sample Size(*n*) for Various Confidence Levels

ConfidenceLevel (%)	Sample Size
95%	16
80%	7
90%	11
97%	19
99%	27
99.9%	43
99.99%	65

Equation

Sample size ***n* = [DEFF*Np(1-p)]/ [(d^2/$Z^2_{1-\alpha/2}$*(N-1) +p*(1-p)]**

$$n = deff \times \frac{N\hat{p}\hat{q}}{\frac{d^2}{1.96^2}(N-1) + \hat{p}\hat{q}}$$

where

n = sample size
$deff$ = design effect
N = population size
$\hat{p}$ = the estimated proportion
$\hat{q} = 1 - \hat{p}$
d = desired absolute precision or absolute level of precision

References

Schaeffer RL, Mendenhall W, Ott L. Elementary Survey Sampling, Fourth Edition. Duxbury Press, Belmont, California 1990.

Methodology

This is a prospective interventional study carried out at Santosh Medical College & Hospital from July 2021-June 2022. This study includes all ages from 18 years and above up to 65 years, all genders, all religions and socioeconomic strata.

A detailed history was collected with particular reference to lower limb ulcers, which side of lower limb, location of ulcer on thigh or leg or foot site & aspect, ulcer onset, duration and type of lesion, location, any associated complaints like varicose veins, Raynaud's phenomena, claudication pain, occupational factors and systemic diseases were taken.

Any history of trauma or similar ulcers in the past were also noted. Any history of smoking or other addictions were taken. A thorough systemic and local examination were carried out. The morphological features of ulcers i.e., Number, distribution of ulcer on thigh or leg or foot site, size, shape, margins, edge, floor, base, discharge, surrounding skin discolouration, and associated diseases like varicose veins, eczema or patches, associated with tenderness, loss of sensations were also noted.

A thorough blood, radiological & histopathological investigations were carried out to support & confirm the diagnosis. This study includes cases of venous ulcers, diabetic ulcers, arterial ulcers, traumatic ulcers and others. Appropriate management according to the cause of ulcer was done. Follow up of 15 days and 2 months in view of healing of ulcer was taken into consideration. But while presenting only relevant positive and some important negative findings were shown to make the study brief and to avoid unnecessary repetitions.

Study Tools:

All the data was recorded and stored in Microsoft excel.

1. History
2. Clinical examination
3. Investigations
    a) Complete blood count
    b) Liver function tests
    c) Fasting and postprandial blood sugar
    d) Serum urea, creatinine
    e) X-ray of local area
    f) USG & doppler of lower limb
    g) Histopathological report of resected specimen (if any)
    h) Pus if found for culture and sensitivity
4. Treatment
5. Follow up- 15 days and 2 months.

Statistical Analysis:

For statistical analysis data were entered into a Microsoft excel spreadsheet and then analysed by SPSS (version 27.0; SPSS Inc., Chicago, IL, USA) and Graph Pad Prism version 5. Data had been summarized as mean and standard deviation for numerical variables and count and percentages for categorical variables.

Z-test (Standard Normal Deviate) was used to test the significant difference of proportions.

Once a t value is determined, a p-value can be found using a table of values from Student's t-distribution. If the calculated p-value is below the threshold chosen for statistical significance (usually the 0.10, the 0.05, or 0.01 level), then the null hypothesis is rejected in favour of the alternative hypothesis. P-value ≤ 0.05 was considered for statistically significant.

RESULTS AND ANALYSIS

RESULT AND ANALYSIS

Table 1:Distribution of Age Groups

Age Groups	Frequency	Percent
≤30	8	12.3%
31-40	7	10.8%
41-50	13	20.0%
51-60	25	38.5%
>60	12	18.5%
Total	65	100.0%

In our study, 8 (12.3%) patients were ≤30 years of age, 7 (10.8%) patients were 31-40 years of age, 13 (20.0%) patient were 41-50 years of age, 25 (38.5%) patients were 51-60 years of age and 12 (18.5%) patients were >60 years of age.

The value of z is 3.6648. The value of p is .00026. The result is significant at p < .05.

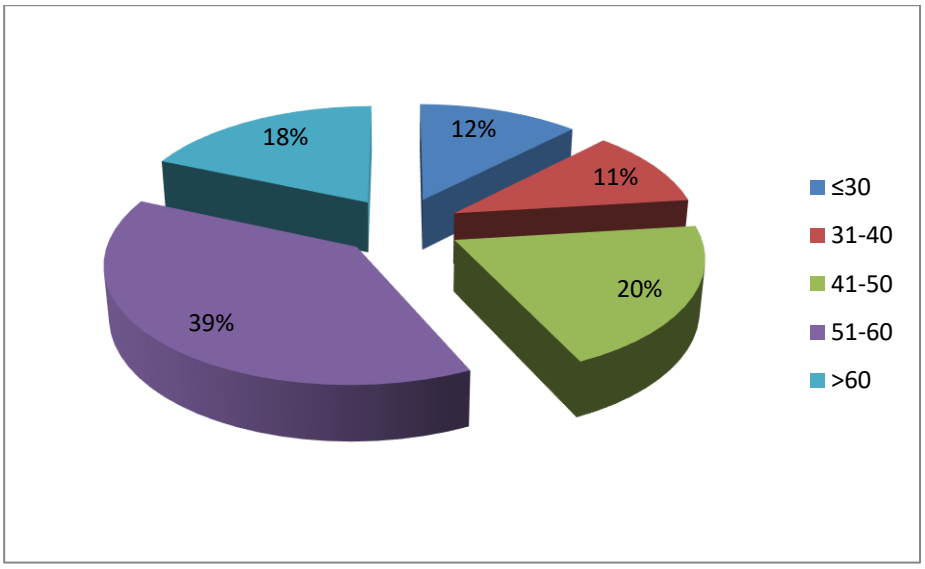


Table 2:: Age group association with Causes of ulcers

	Causes of ulcers				
Age group	Arterial	Diabetes	Trauma	Venous	Grand Total
≤30	3	1	1	3	8
≥61	4	4	0	4	12
31-40	1	2	1	3	7
41-50	2	3	1	7	13
51-60	1	12	3	9	25
Grand Total	11	22	6	26	65

In our study out of 8 patients in age group <30 years arterial and venous cause ulcers are most common, out of 7 patients in age group 31-40 years venous cause of ulcers are most common, out of 13 patients in age group 41-50 venous cause of ulcers are most common, out of 25 patients in age group 51-60 years diabetic cause of ulcers are most common followed by venous cause, out of 12 patients in age group > 61 years arterial, venous, diabetic cause of ulcers are equal in number.

The value of z is 13.289. The value of p is .001. The result is significant at p < .05.

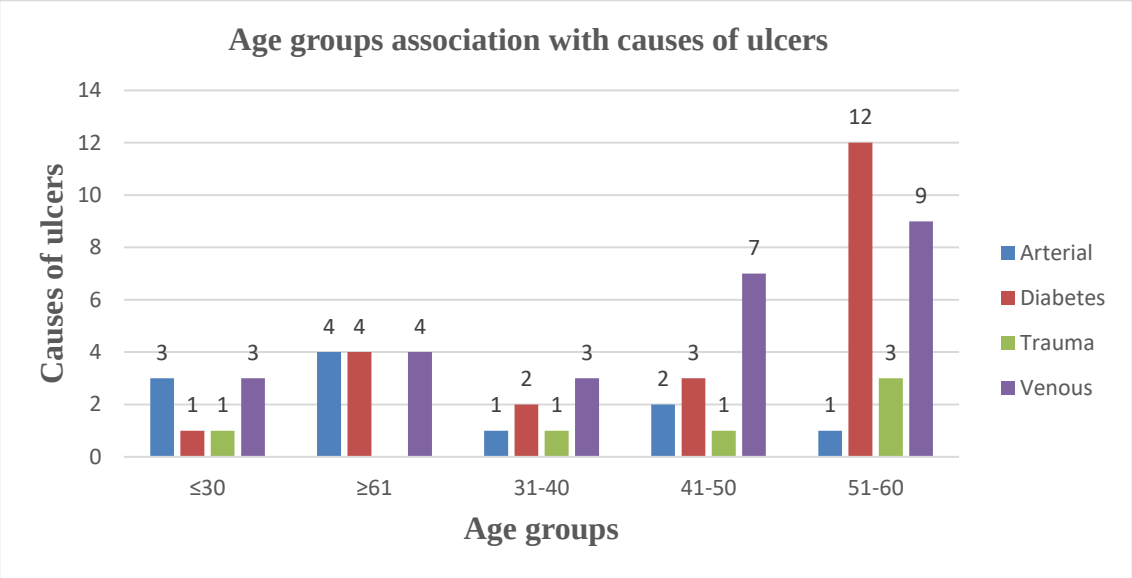


Table 3: Distribution of Gender

Gender	Frequency	Percent
Female	20	30.8%
Male	45	69.2%
Total	65	100.0%

In our study, 20 (30.8%) patients were Female and 45 (69.2%) patients were Male.

The value of z is 4.3853. The value of p is < .00001. The result is significant at p < .05.

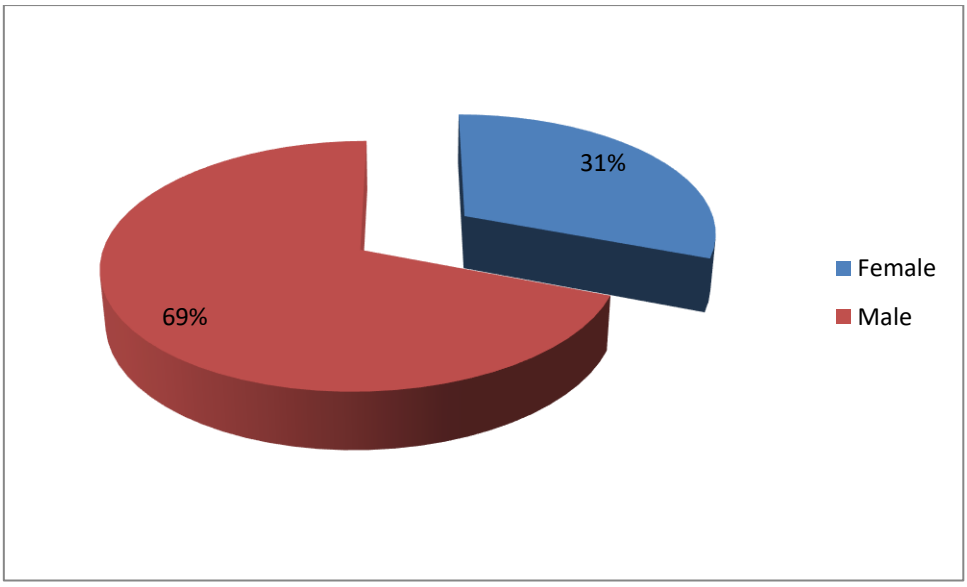


Table 4:Distribution of Life Style

Life Style	Frequency	Percent
Heavy Work	18	27.7%
Moderate	14	21.5%
Sedentary	33	50.8%
Total	65	100.0%

In our study, 18 (27.7%) patients had Heavy Work, 14 (21.5%) patients had Moderate and 33 (50.8%) patients had Sedentary Life Style.

The value of z is 3.4685. The value of p is .00052. The result is significant at $p < .05$.

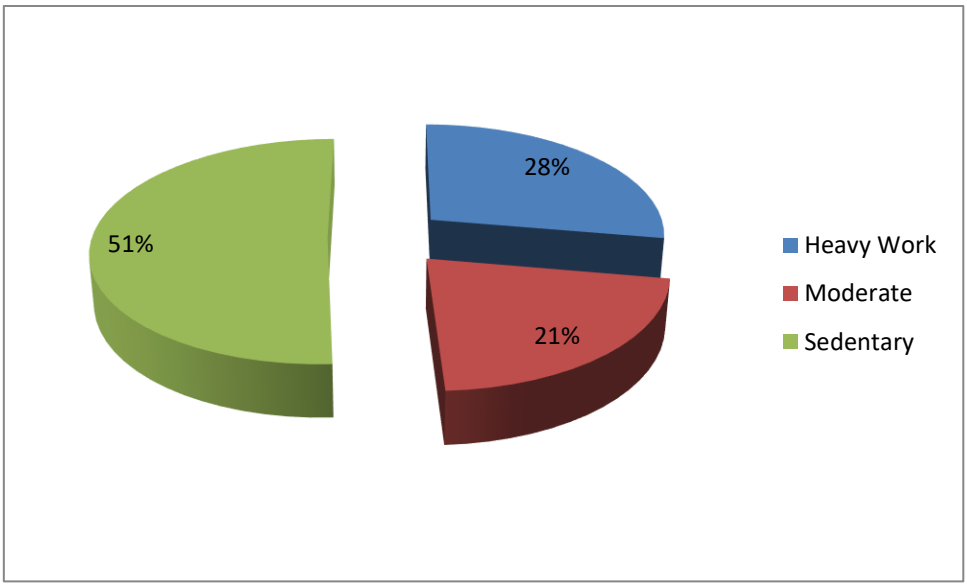


Table 5: Distribution of Location of Ulcer

Location Of Ulcer	Frequency	Percent
Foot	28	43.1%
Leg	34	52.3%
Thigh	3	4.6%
Total	65	100.0%

In our study, 28 (43.1%) patients had Foot, 34 (52.3%) patients had Leg and 3 (4.6%) patients had Thigh in Location of Ulcer.

The value of z is 6.0255. The value of p is < .00001. The result is significant at p < .05.

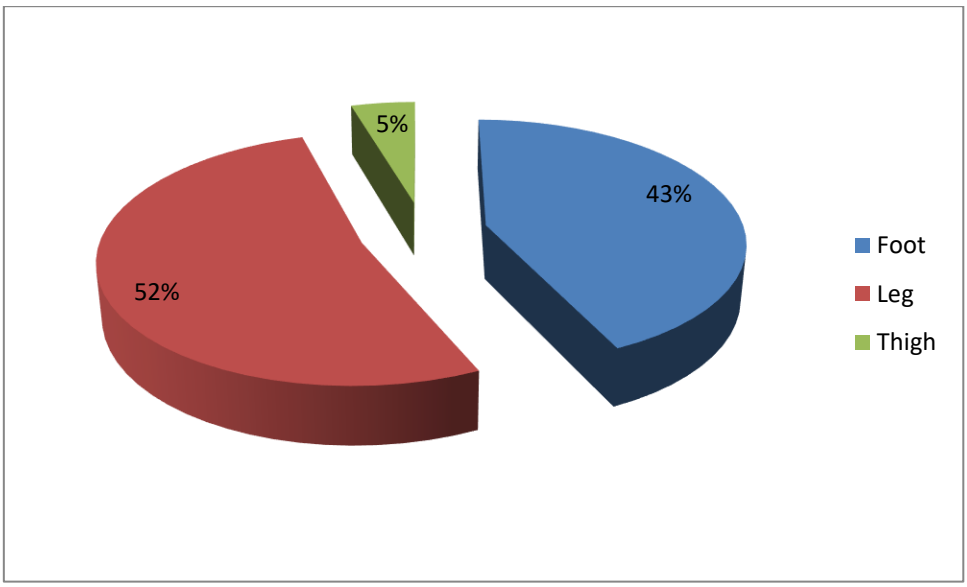


Table 6: Distribution of Aspect

Aspect	Frequency	Percent
Anterior	4	6.2%
Dorsal	17	26.2%
Lateral	18	27.7%
Medial	16	24.6%
Plantar	5	7.7%
Posterior	5	7.7%
Total	65	100.0%

In our study, 4 (6.2%) patients had Anterior, 17 (26.2%) patients had Dorsal and 18 (27.2%) patients had Lateral, 18 (27.7%) patients had Lateral, 16 (24.6%) patients had Medial, 5 (7.7%) patients had Plantar, 5 (7.7%) patients had Posterior.

The value of z is 3.2747. The value of p is .00108. The result is significant at $p < .05$.

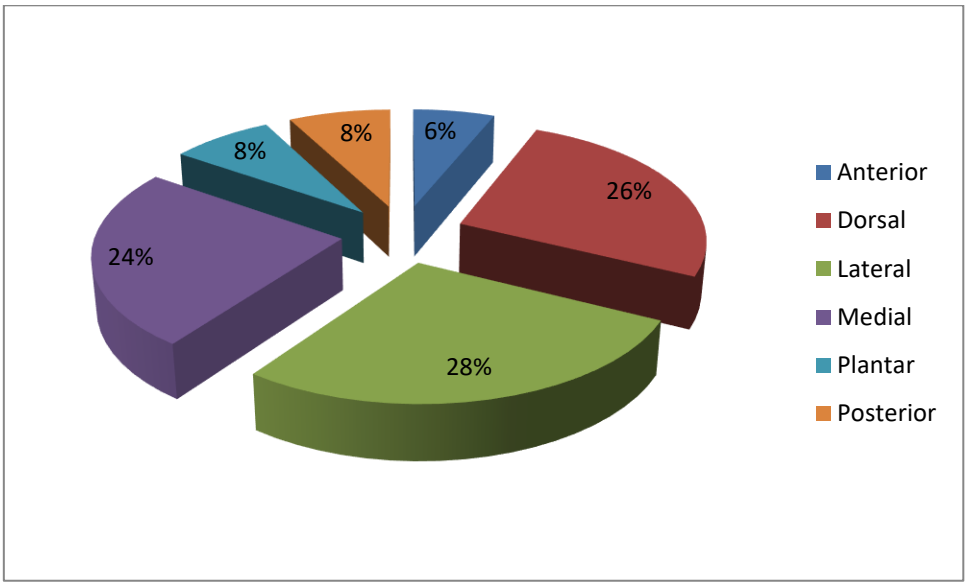


Table 7: Distribution of Side

Side	Frequency	Percent
Left	42	64.6%
Right	23	35.4%
Total	65	100.0%

In our study, 42 (64.6%) patients had Left Side and 23 (35.4%) patients were Right Side.

The value of z is 3.3328. The value of p is .00086. The result is significant at $p < .05$.

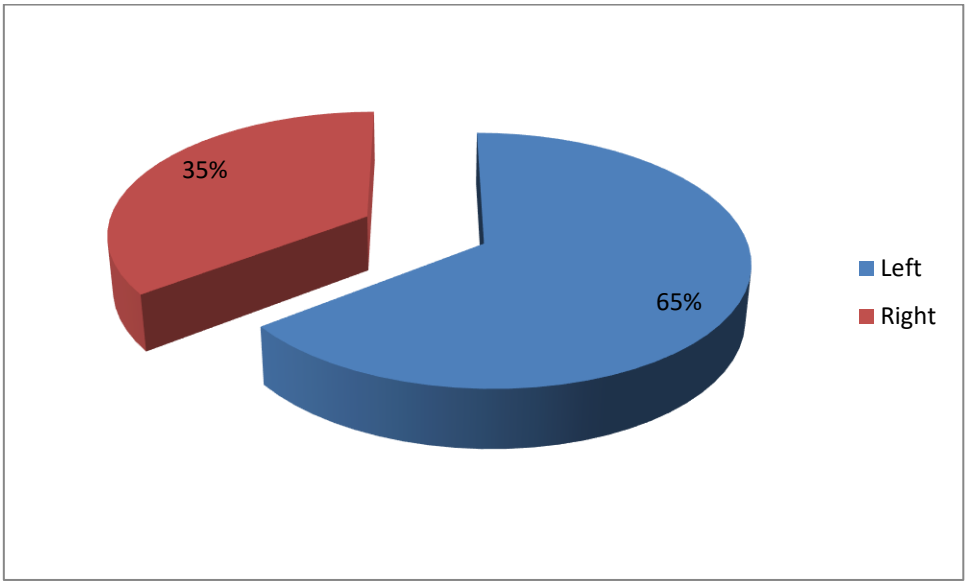


Table 8: Distribution of H/O Trauma

H/O Trauma	Frequency	Percent
No	47	72.3%
Yes	18	27.7%
Total	65	100.0%

In our study, 18 (27.7%) patients had H/O Trauma.

The value of z is 5.0869. The value of p is < .00001. The result is significant at p < .05.

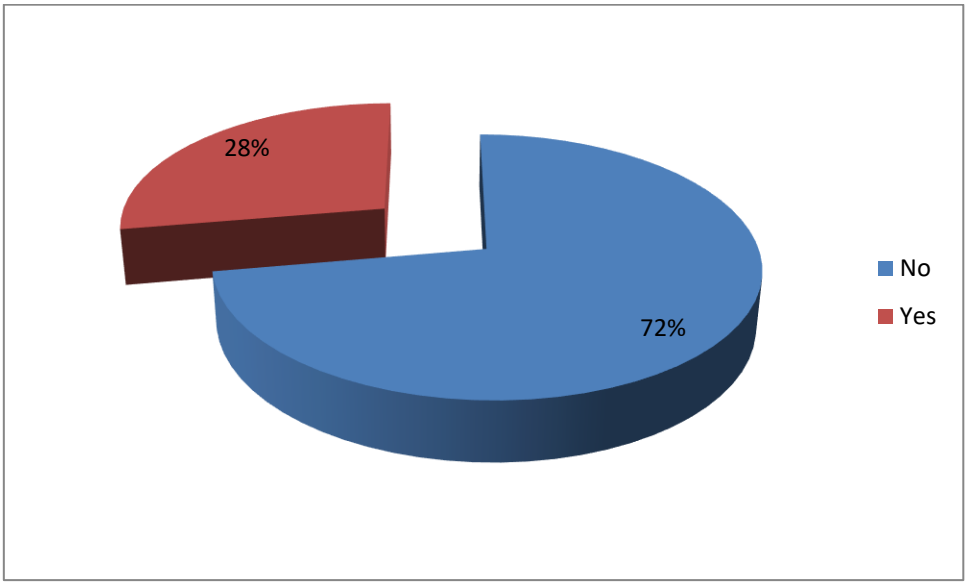


Table 9: Distribution of Varicose Veins

Varicose Veins	Frequency	Percent
Absent	57	87.7%
Present	8	12.3%
Total	65	100.0%

In our study, 8 (12.3%) patients had Varicose Veins.

The value of z is 8.5952. The value of p is < .00001. The result is significant at p < .05.

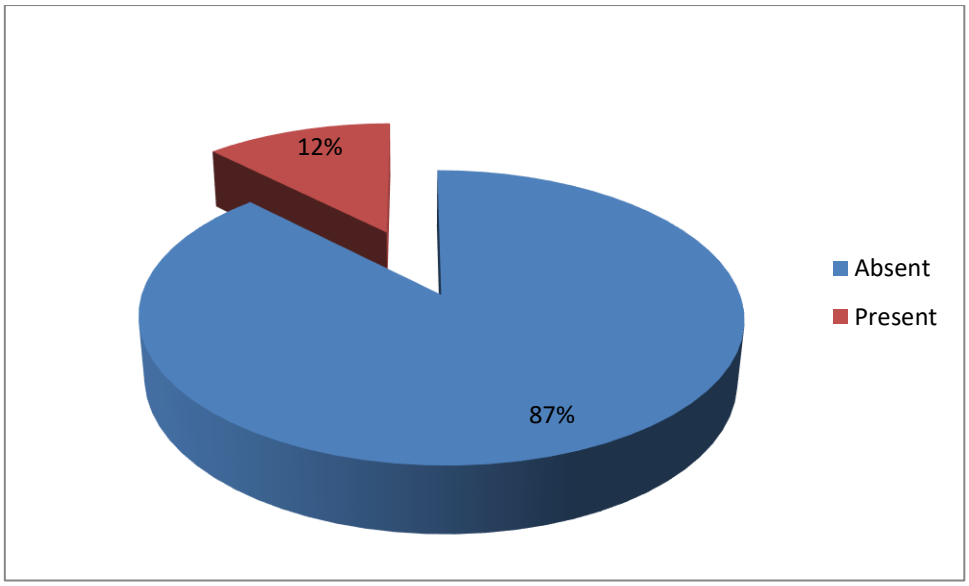


Table 10: Causes of ulcers association with Varicose veins

	Varicose veins		
Causes of ulcers	Absent	Present	Grand Total
Arterial	11	0	11
Diabetes	22	0	22
Trauma	6	0	6
Venous	18	8	26
Grand Total	57	8	65

In our study out of 8 patients presented with varicose veins the cause of ulcers is of venous aetiology

The value of z is 1.002. The value of p is < .001. The result is significant at p < .05.

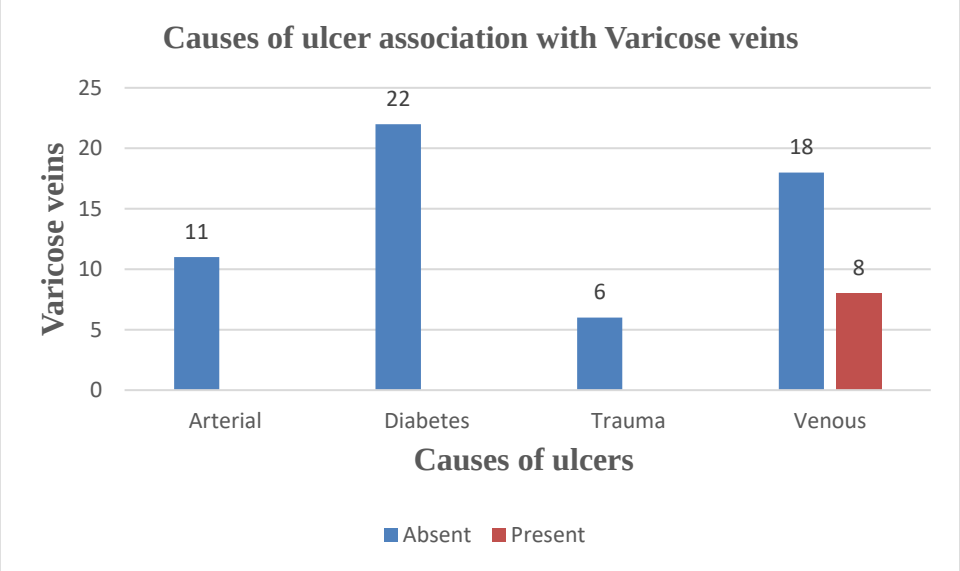


Table 11: Distribution of Claudication Pain

Claudication Pain	Frequency	Percent
No	57	87.7%
Yes	8	12.3%
Total	65	100.0%

In our study, 8 (12.3%) patients had Claudication Pain.

The value of z is 8.5952. The value of p is < .00001. The result is significant at p < .05.

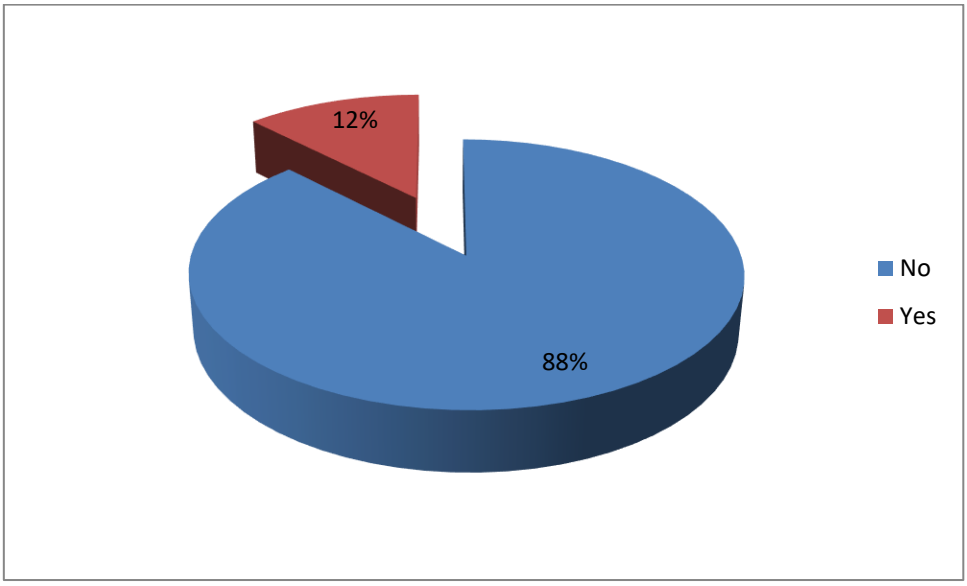


Table 12: Distribution of Raynaud's Phenomena

Raynaud's Phenomena	Frequency	Percent
No	60	92.3%
Yes	5	7.7%
Total	65	100.0%

In our study, 5 (7.7%) patients had Raynaud's Phenomena.

The value of z is 9.6476. The value of p is < .00001. The result is significant at p < .05.

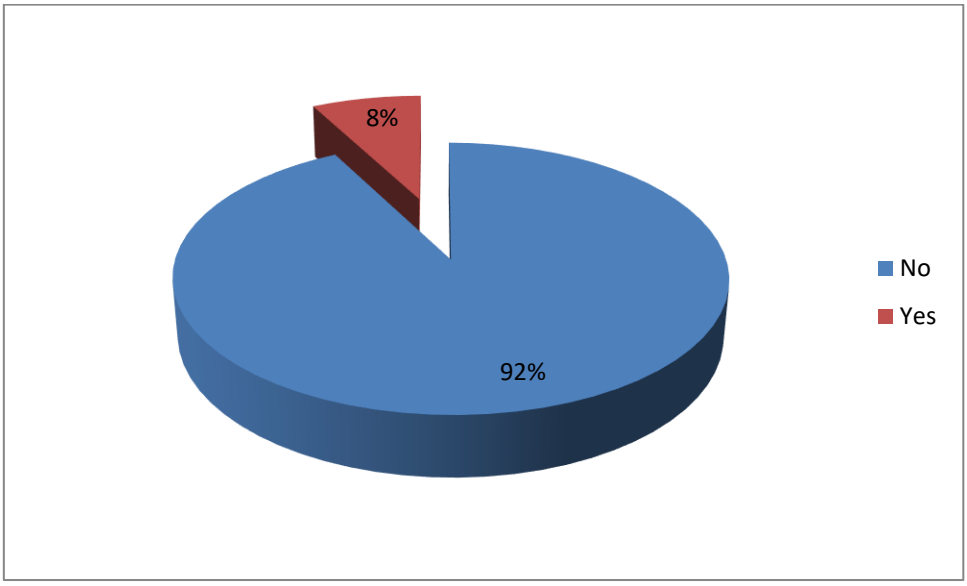


Table 13: Causes of ulcers association with Raynaud's phenomena

	Raynaud's phenomena		
Causes of ulcers	Absent	Present	Grand Total
Arterial	6	5	11
Diabetes	22	0	22
Trauma	6	0	6
Venous	26	0	26
Grand Total	60	5	65

In our study 5 patients had Raynaud's phenomena and the cause of ulcers are of arterial aetiology.

The value of z is 0.899. The value of p is < .001. The result is significant at p < .05.

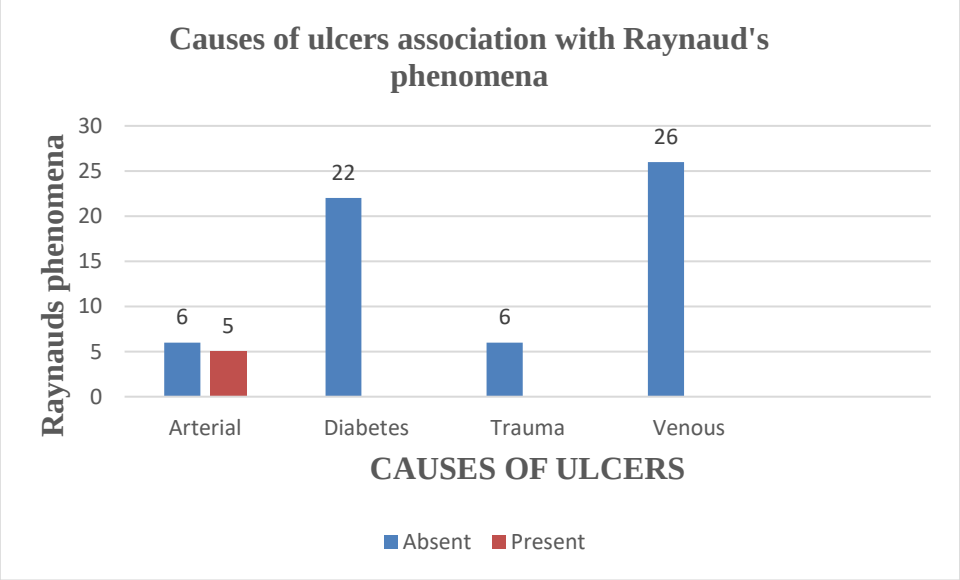


Table 14: Distribution of Loss of Sensation

Loss Of Sensation	Frequency	Percent
No	44	67.7%
Yes	21	32.3%
Total	65	100.0%

In our study, 21 (32.3%) patients had Loss of Sensation.

The value of z is 4.0345. The value of p is < .00001. The result is significant at p < .05.

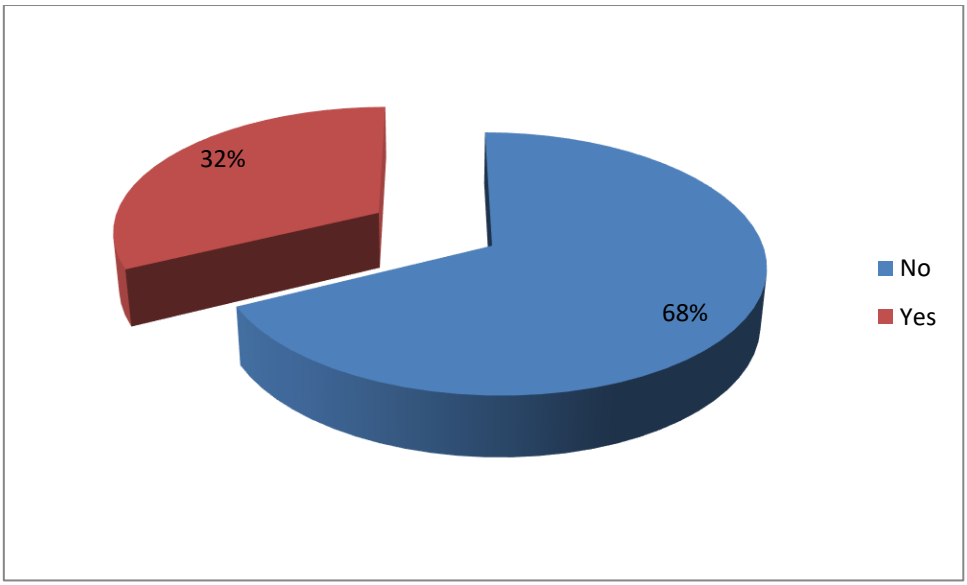


Table 15: Causes of ulcers association with loss of sensation

	Loss of sensation		
Causes of ulcers	Absent	Present	Grand Total
Arterial	4	7	11
Diabetes	14	8	22
Trauma	6	0	6
Venous	20	6	26
Grand Total	44	21	65

In our study out of 21 patients with loss of sensation the causes of ulcers are diabetic followed by arterial and venous aetiology.

The value of z is 1.074. The value of p is < .001. The result is significant at p < .05.

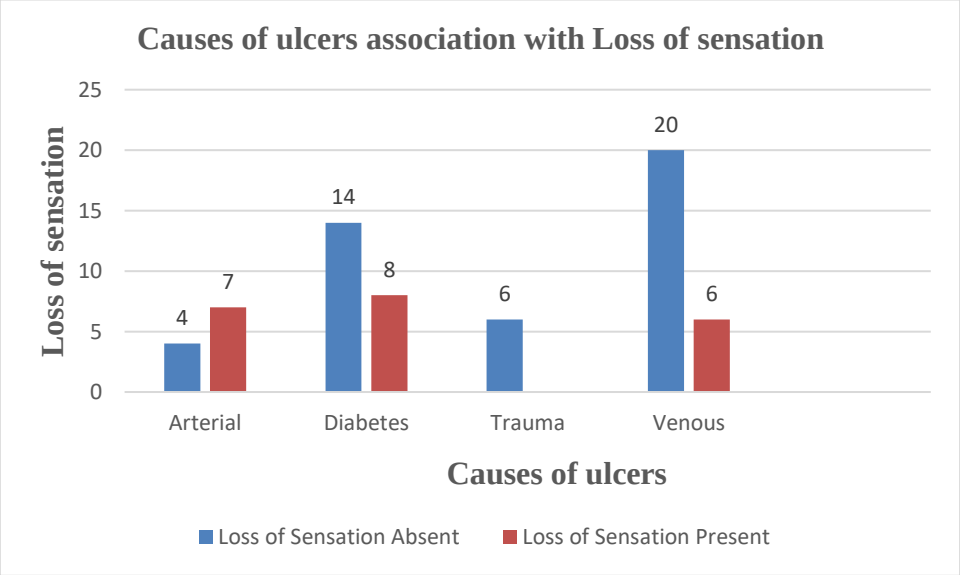


Table 16: Distribution of Peripheral Pulses

Peripheral Pulses	Frequency	Percent
Absent	6	9.2%
Present	59	90.8%
Total	65	100.0%

In our study, 59 (90.8%) patients had Peripheral Pulses.

The value of z is 9.2968. The value of p is < .00001. The result is significant at p < .05.

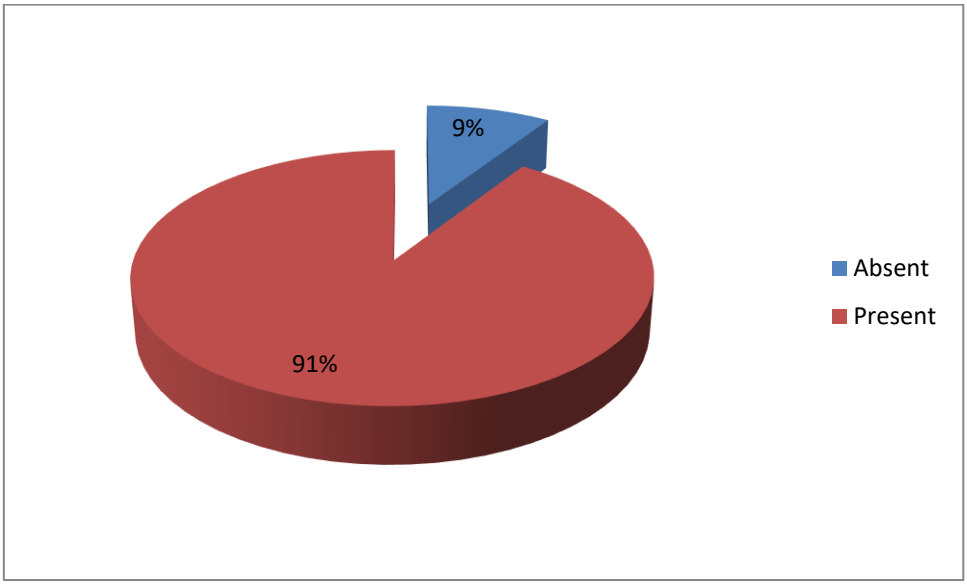


Table 17: Causes of ulcers association with Peripheral pulses

	Peripheral pulses		
Causes of ulcers	Absent	Present	Grand Total
Arterial	5	6	11
Diabetes	0	22	22
Trauma	0	6	6
Venous	1	25	26
Grand Total	6	59	65

In our study 6 patients had absent peripheral pulses and the causes of ulcers are arterial followed by venous aetiology.

The value of z is 1.062. The value of p is < .001. The result is significant at p < .05.

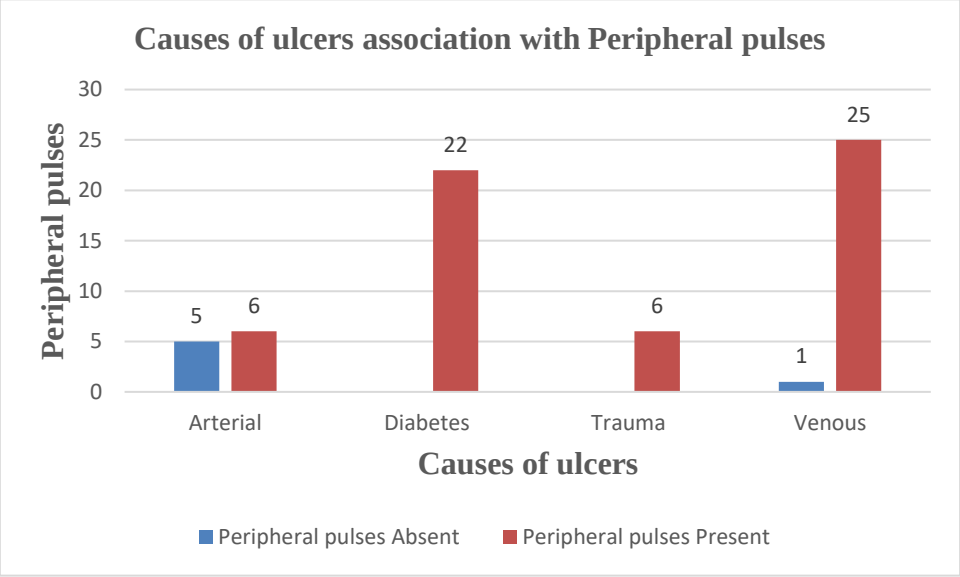


Table 18: Distribution of HTN

HTN	Frequency	Percent
No	38	58.5%
Yes	27	41.5%
Total	65	100.0%

In our study, 27 (41.5%) patients had HTN.

The value of z is 1.9295. The value of p is .0536. The result is not significant at p < .05.

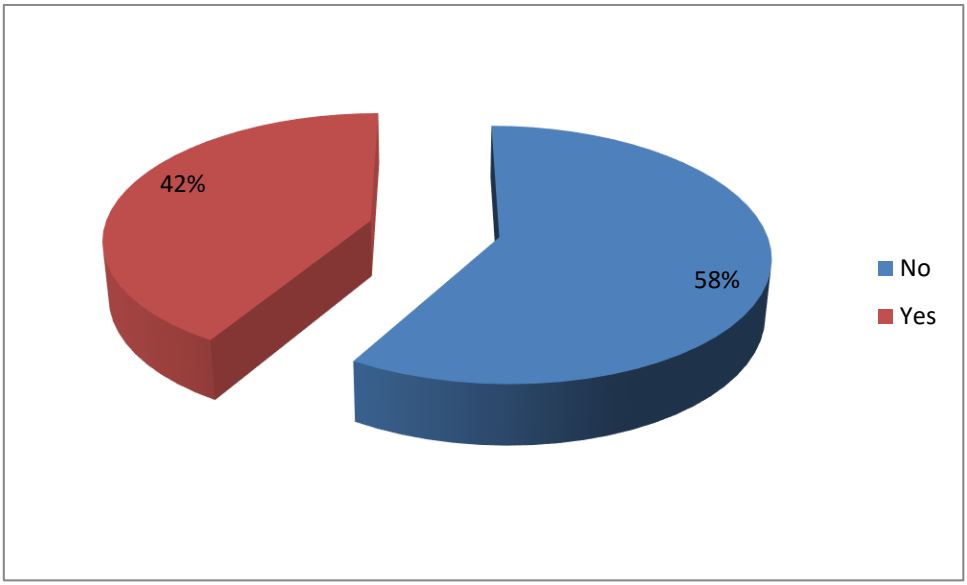


Table 19: Causes of ulcers association with HTN

	HTN		
Causes of ulcers	Absent	Present	Grand Total
Arterial	3	8	11
Diabetes	13	9	22
Trauma	4	2	6
Venous	18	8	26
Grand Total	38	27	65

In our study 27 patients had HTN in them causes of ulcers are diabetic followed by equal number of arterial and venous aetiology.

The value of z is 1.028. The value of p is .001. The result is significant at $p < .05$

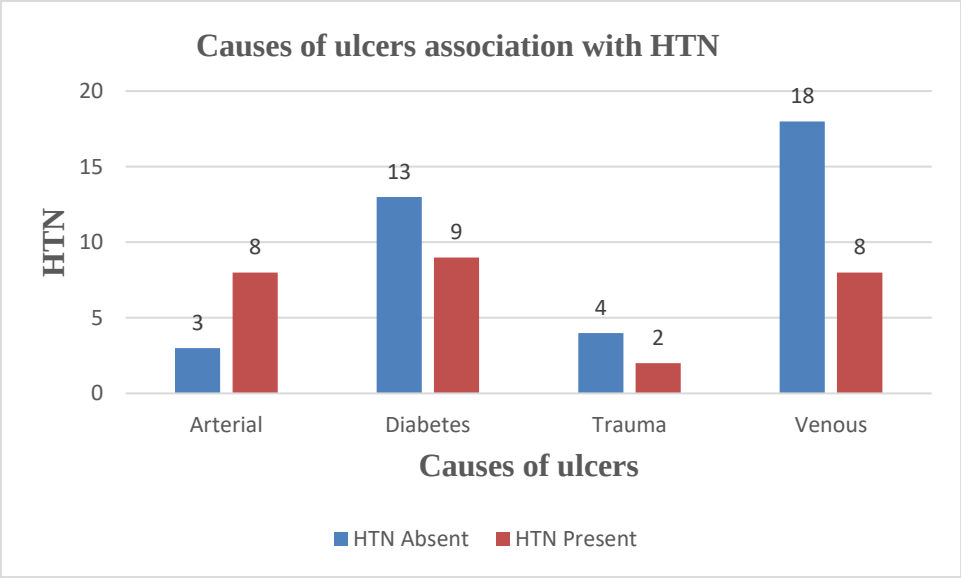


Table 20: Distribution of DM

DM	Frequency	Percent
No	26	40.0%
Yes	39	60.0%
Total	65	100.0%

In our study, 39 (60.0%) patients had DM.

The value of z is 2.2804. The value of p is .0226. The result is significant at p < .05.

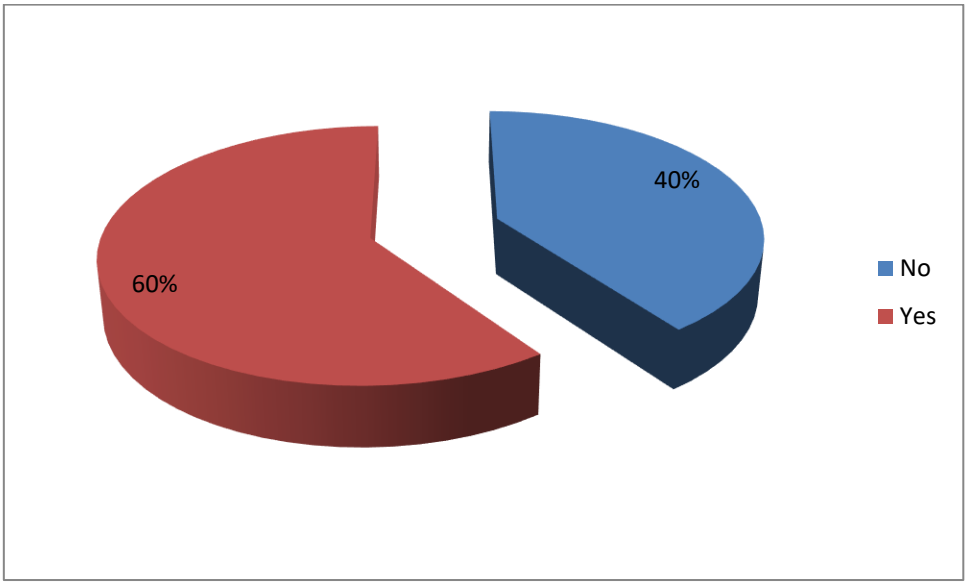


Table 21: Causes of ulcers association with DM

	DM		
Causes of ulcers	Absent	Present	Grand Total
Arterial	6	5	11
Diabetes	0	22	22
Trauma	6	0	6
Venous	14	12	26
Grand Total	26	39	65

In our study 39 patients had DM among them the causes of ulcers are diabetic followed by venous and arterial aetiology.

The value of z is 1.298. The value of p is .001. The result is significant at $p < .05$.

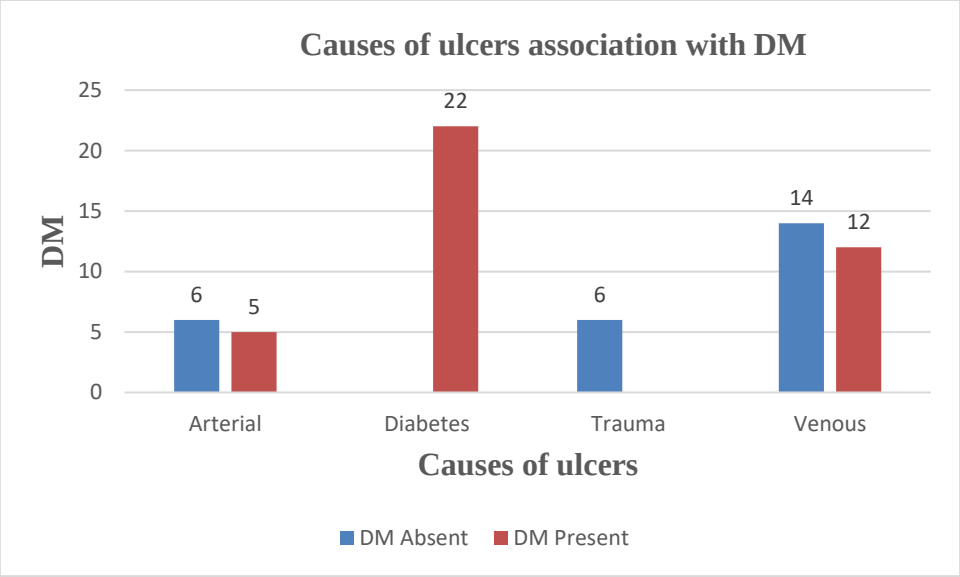


Table 22: Distribution of H/O Smoking

H/O Smoking	Frequency	Percent
No	21	32.3%
Yes	44	67.7%
Total	65	100.0%

In our study, 44 (67.7%) patients were H/O Smoker.

The value of z is 4.0345. The value of p is < .00001. The result is significant at p < .05.

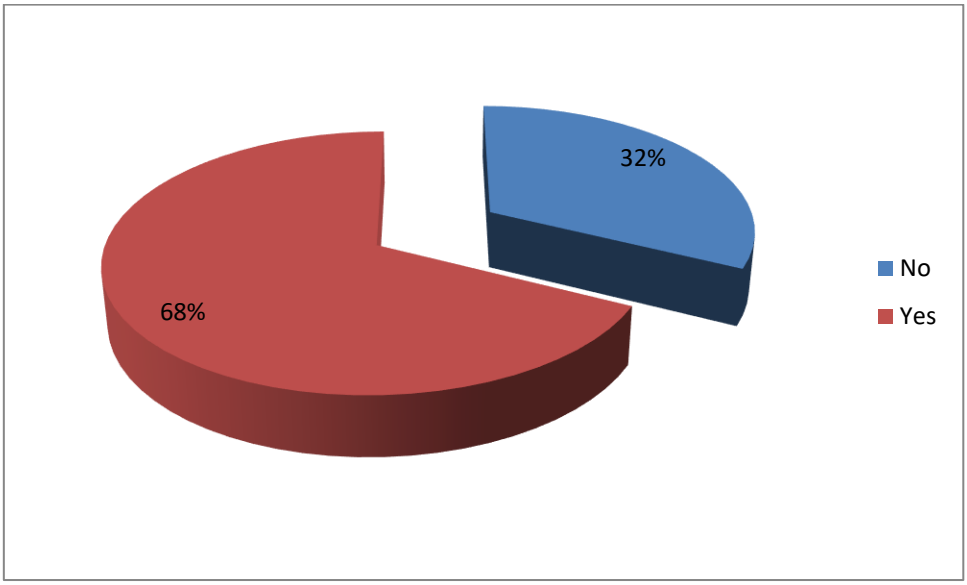


Table 23: Causes of ulcer association with H/O Smoking

	H/O Smoking		
Causes of ulcers	Absent	Present	Grand Total
Arterial	1	10	11
Diabetes	5	17	22
Trauma	2	4	6
Venous	13	13	26
Grand Total	21	44	65

In our study 44 patients had H/O smoking among them the causes of ulcers are diabetic followed by venous and arterial aetiology.

The value of z is 1.050. The value of p is < .001. The result is significant at p < .05.

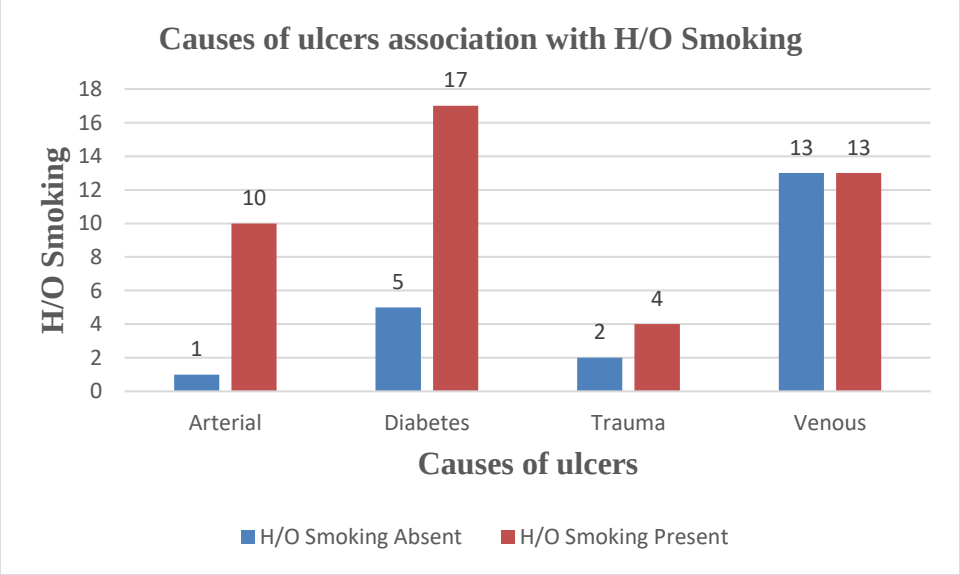


Table 24: Distribution of Edges

Edges	Frequency	Percent
Everted	2	3.1%
Punched Out	32	49.2%
Slopping	29	44.6%
Undermined	2	3.1%
Total	65	100.0%

In our study, 2 (3.1%) patients had averted, 32 (49.2%) patients had Punched Out, 29 (44.6%) patients had Slopping, 2 (3.1%) patients had undermined.

The value of z is 5.9871. The value of p is < .00001. The result is significant at p < .05.

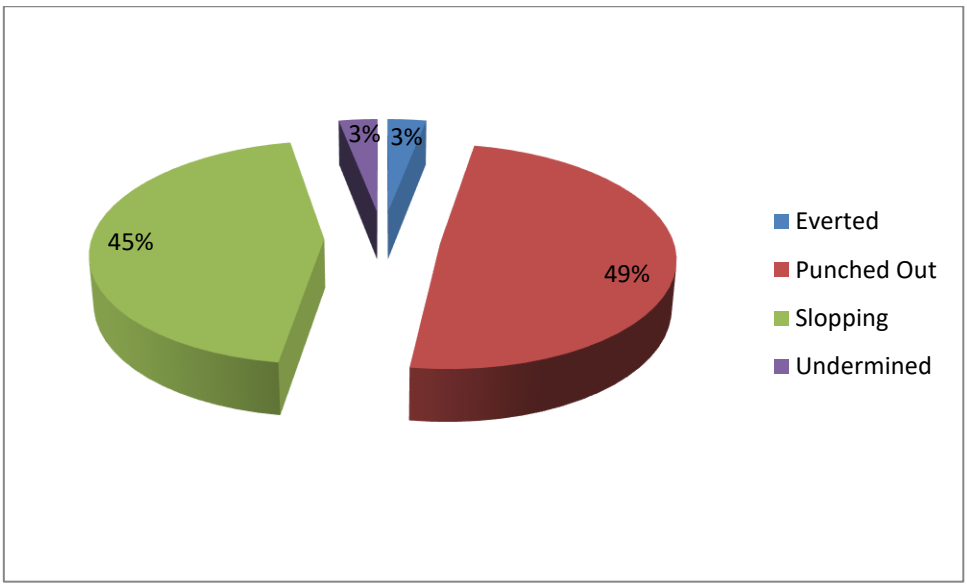


Table 25: Distribution of Margin

Margin	Frequency	Percent
Irregular	26	40.0%
Regular	39	60.0%
Total	65	100.0%

In our study, 26 (40.0%) patients had Irregular Margin, 39 (60.0%) patients had Regular Margin.

The value of z is 2.2804. The value of p is .0226. The result is significant at p < .05.

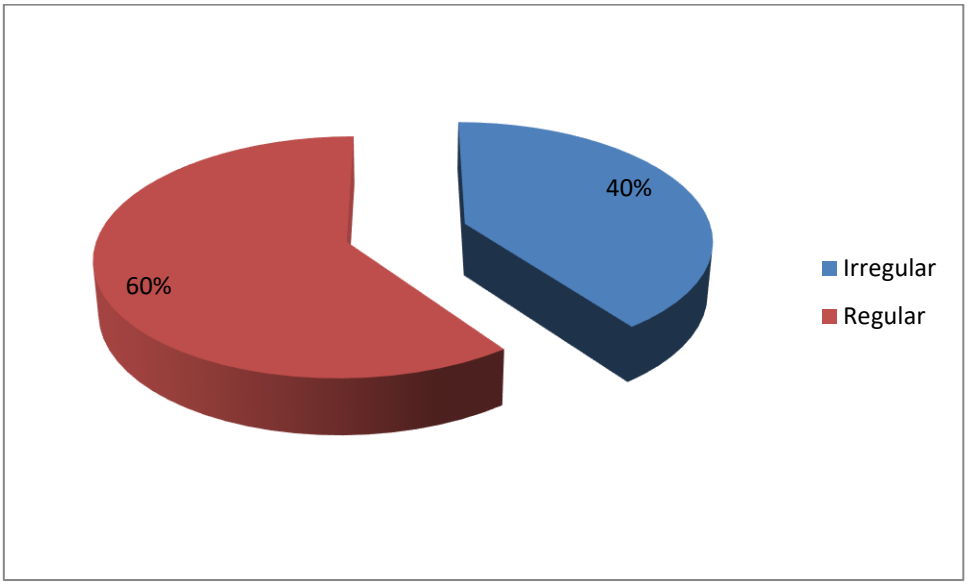


Table 26: Distribution of Floor

Floor	Frequency	Percent
Granulation Tissue	33	50.8%
Slough	32	49.2%
Total	65	100.0%

In our study, 33 (50.8%) patients had Granulation Tissue, 32 (49.2%) patients had Slough in Floor.

The value of z is 0.1754. The value of p is .85716. The result is not significant at p < .05.

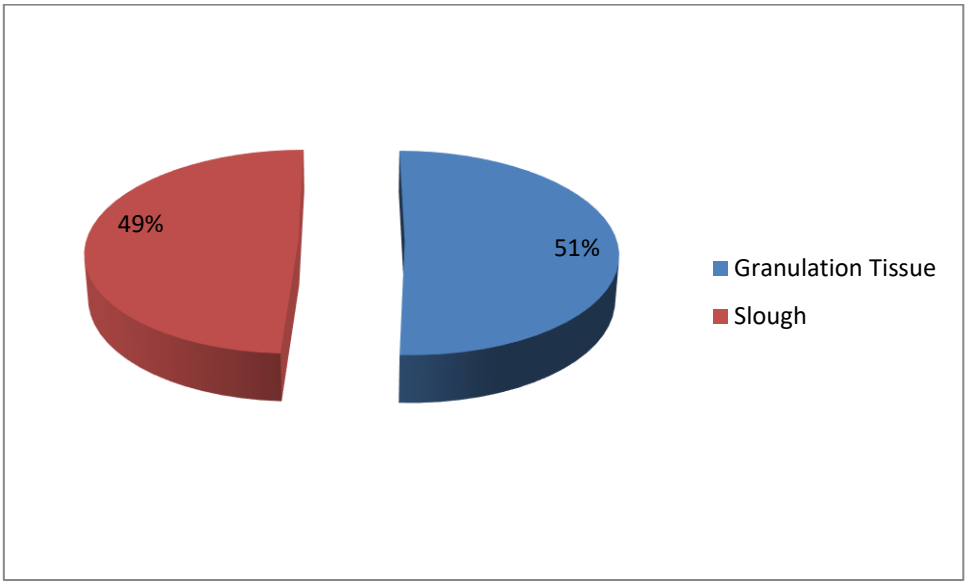


Table 27: Distribution of Base

Base	Frequency	Percent
Bone	35	53.8%
Muscle	15	23.1%
Tendons	15	23.1%
Total	65	100.0%

In our study, 35 (53.8%) patients had Bone, 15 (23.1%) patients had Muscle and 15 (23.1%) patients had Tendons.

The value of z is 3.6056. The value of p is .0003. The result is significant at $p < .05$.

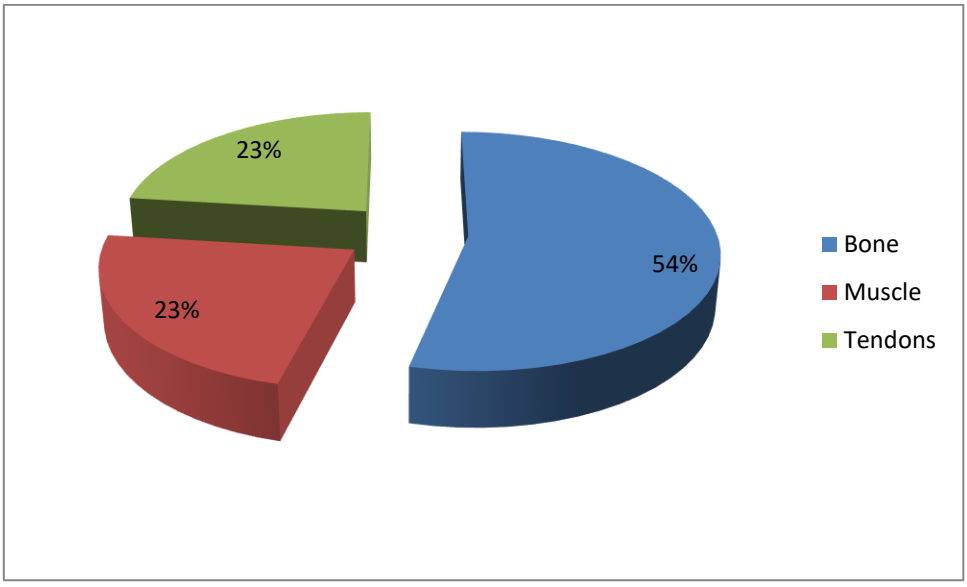


Table 28: Distribution of Surrounding Skin Discolouration

Surrounding Skin Discolouration	Frequency	Percent
No	36	55.4%
Yes	29	44.6%
Total	65	100.0%

In our study, 29 (44.6%) patients had Surrounding Skin Discolouration.

The value of z is 1.2279. The value of p is .2187. The result is not significant at p < .05.

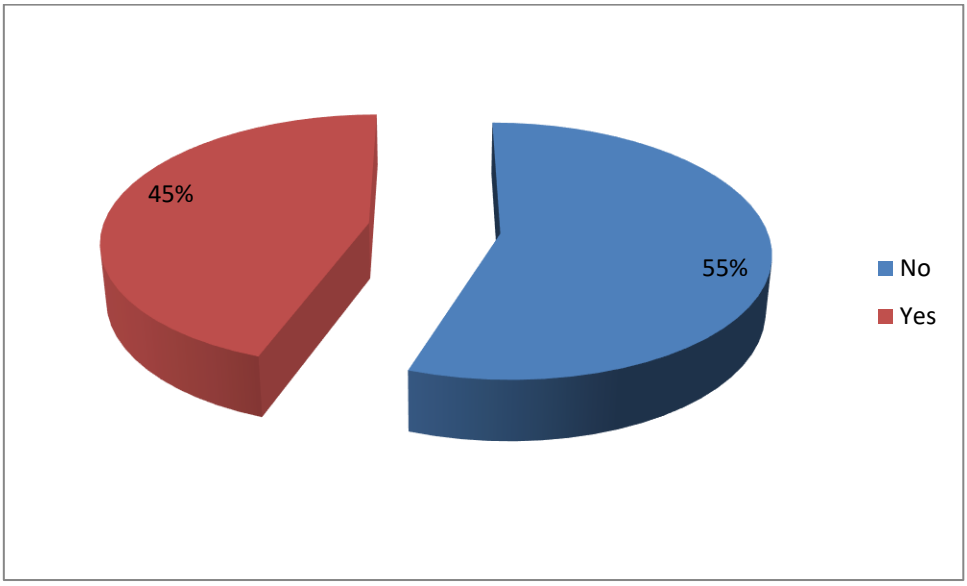


Table 29: Distribution of Discharge

Discharge	Frequency	Percent
No	38	58.5%
Yes	27	41.5%
Total	65	100.0%

In our study, 27 (41.5%) patients had Discharge.

The value of z is 1.9295. The value of p is .0536. The result is not significant at p < .05.

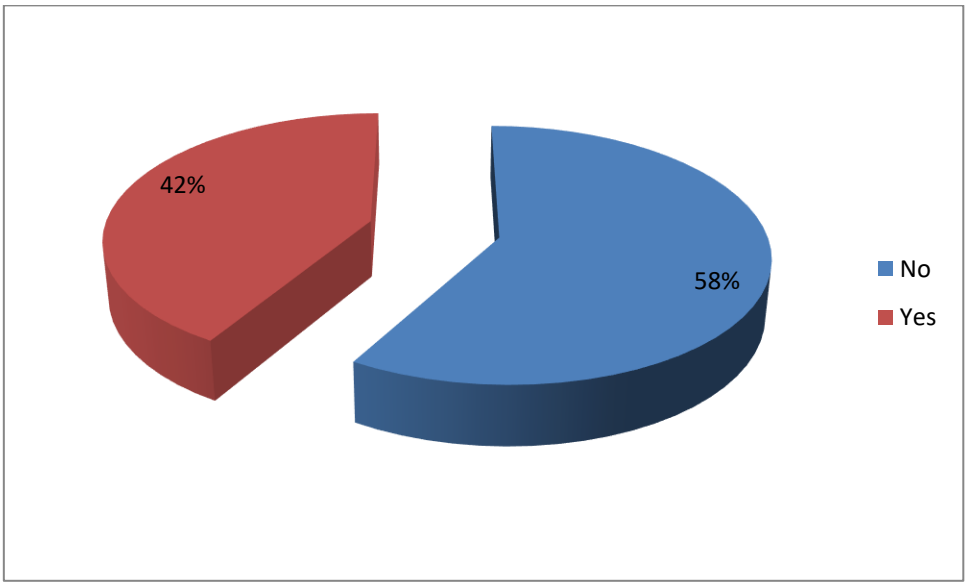


Table 30: Distribution of Culture (Organism)

Culture (Organism)	Frequency	Percent
Growth Seen	35	53.8%
Sterile	30	46.2%
Total	65	100.0%

In our study, 35 (53.8%) patients had Growth Seen, 30 (46.2%) patients had Sterile in Culture (Organism).

The value of z is 0.8771. The value of p is .37886. The result is not significant at p < .05.

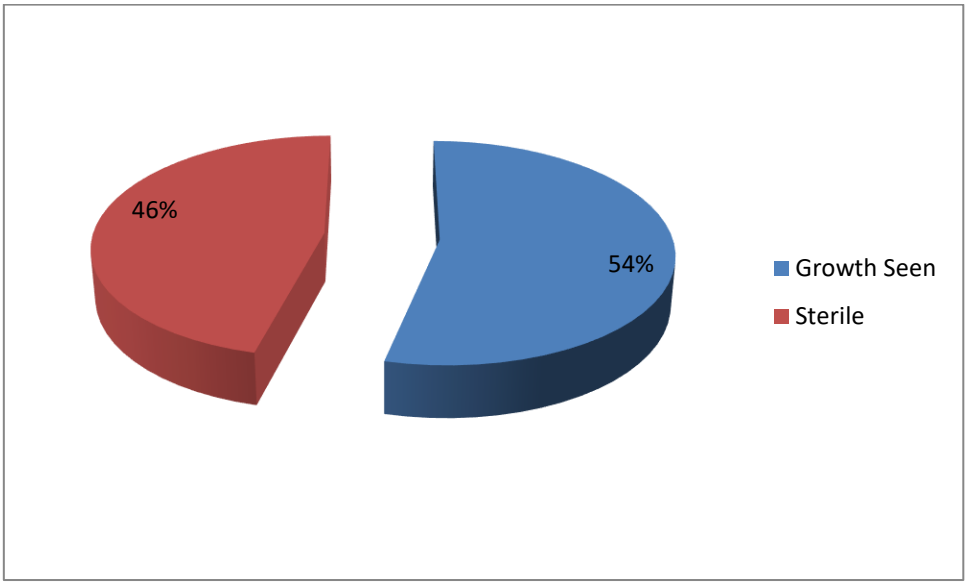


Table 31: Distribution of USG Colour Doppler Study

USG Colour Doppler Study	Frequency	Percent
Arterial System Involved	11	16.9%
Ns	37	56.9%
Venous System Involved	17	26.2%
Total	65	100.0%

In our study, 11 (16.9%) patients had Arterial System Involved, 37 (56.9%) patients had Ns, 17 (26.2%) patients had Venous System Involved.

The value of z is 4.7252. The value of p is < .00001. The result is significant at p < .05.

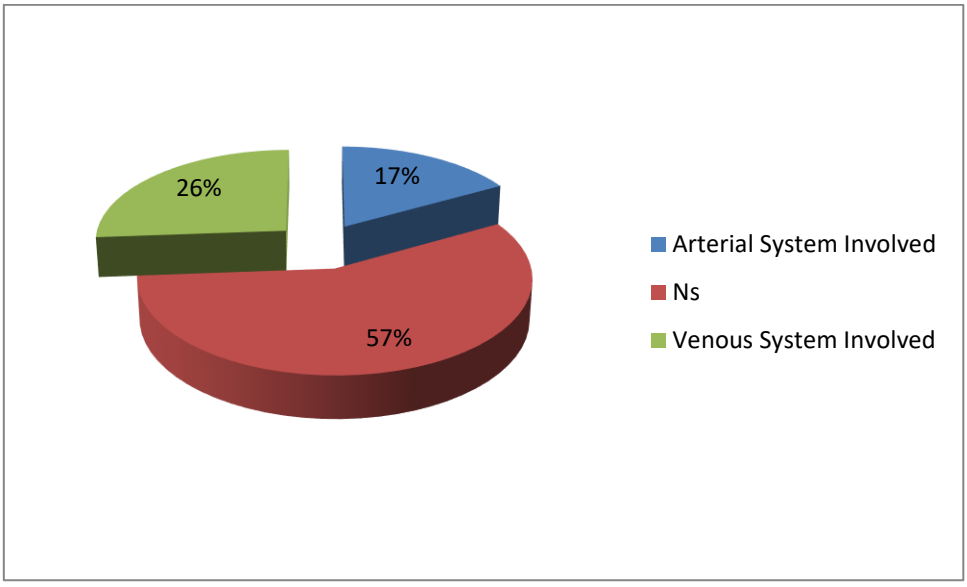


Table 32: Distribution of X-RAY

X-RAY	Frequency	Percent
Bone involved	6	9.2%
NAD	59	90.8%
Total	65	100.0%

In our study, 6 (9.2%) patients had Bone involved, 59 (90.8%) patients had NAD.

The value of z is 9.2968. The value of p is < .00001. The result is significant at p < .05.

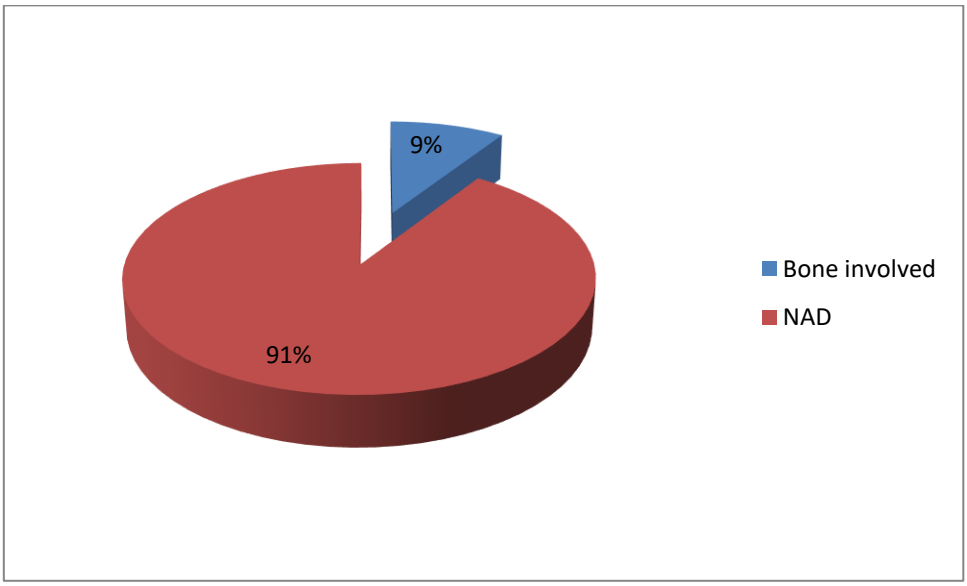


Table 33: Distribution of Biopsy

Biopsy	Frequency	Percent
NAD	63	96.9%
Premalignant Growth	2	3.1%
Total	65	100.0%

In our study, 63 (96.9%) patients had NAD, 2 (3.1%) patients had Premalignant Growth.

The value of z is 10.7001. The value of p is < .00001. The result is significant at p < .05.

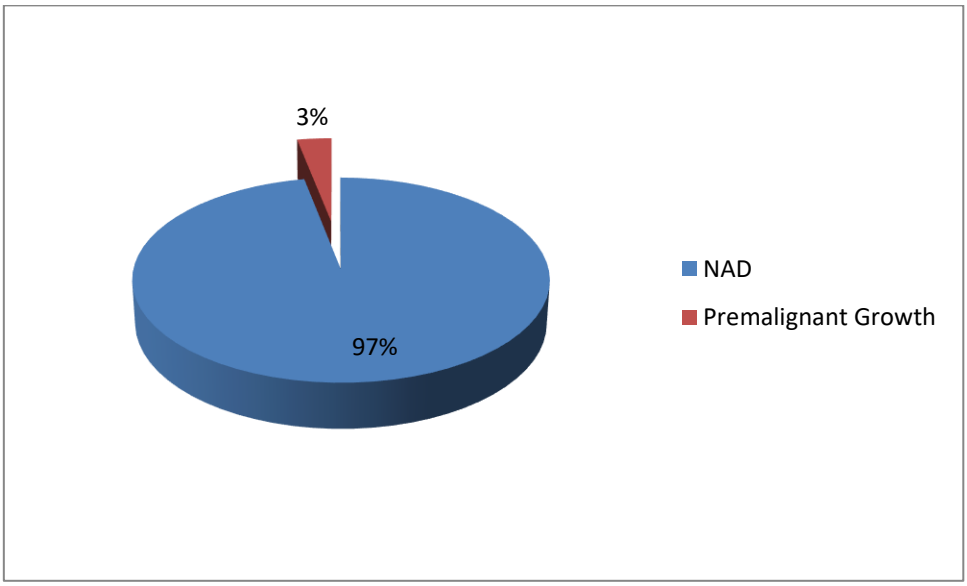
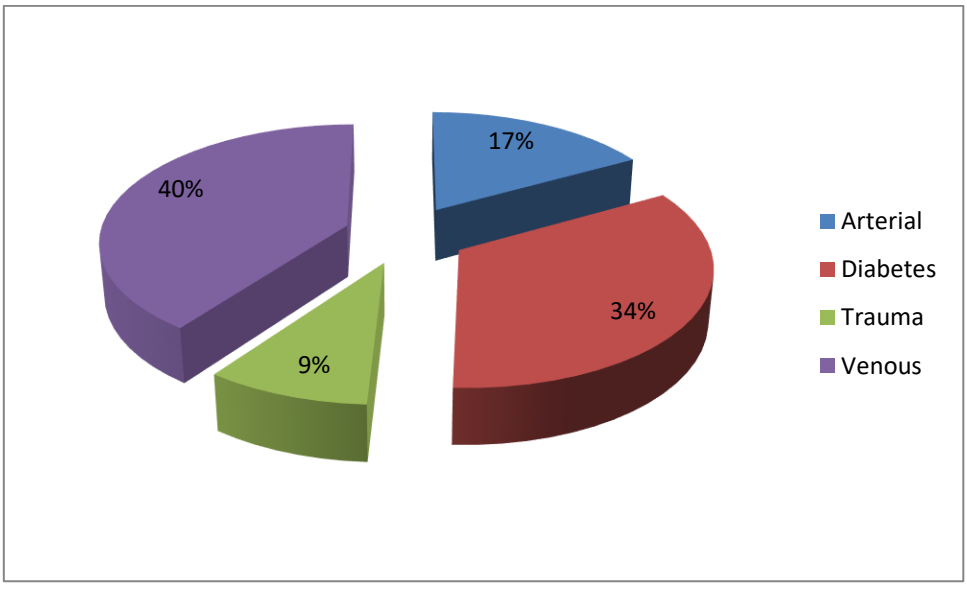


Table 34: Distribution of Cause of Ulcer

Cause Of Ulcer	Frequency	Percent
Arterial	11	16.9%
Diabetes	22	33.8%
Trauma	6	9.2%
Venous	26	40.0%
Total	65	100.0%

In our study, 11 (16.9%) patients had Arterial, 22 (33.8%) patients had Diabetes, 6 (9.2%) patients had Diabetes and 26 (40.0%) patients had Venous.

The value of z is 4.0721. The value of p is < .00001. The result is significant at p < .05.



According to Gilliland 95% of leg ulcers are due to vascular etiology and venous ulcers dominates accounting for up 90% of the cases. Arterial ulcers account for 5 & 10% and others are due to neuropathy or a combination of both. (Young RJ)[16]

Table 35: Distribution of Conservative

Conservative	Frequency	Percent
No	55	84.6%
Yes	10	15.4%
Total	65	100.0%

In our study, 10 (15.4%) patients had Conservative.

The value of z is 7.8935. The value of p is < .00001. The result is significant at p < .05.

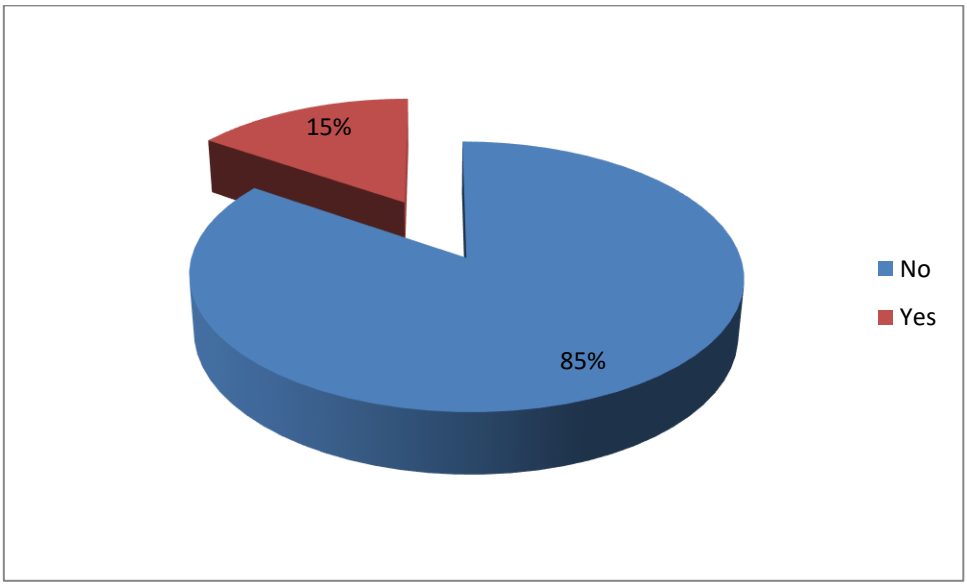


Table 36: Distribution of Surgical Debridement

Surgical Debridement	Frequency	Percent
No	36	55.4%
Yes	29	44.6%
Total	65	100.0%

In our study, 29 (44.6%) patients had Surgical Debridement.

The value of z is 1.2279. The value of p is .2187. The result is not significant at p < .05.

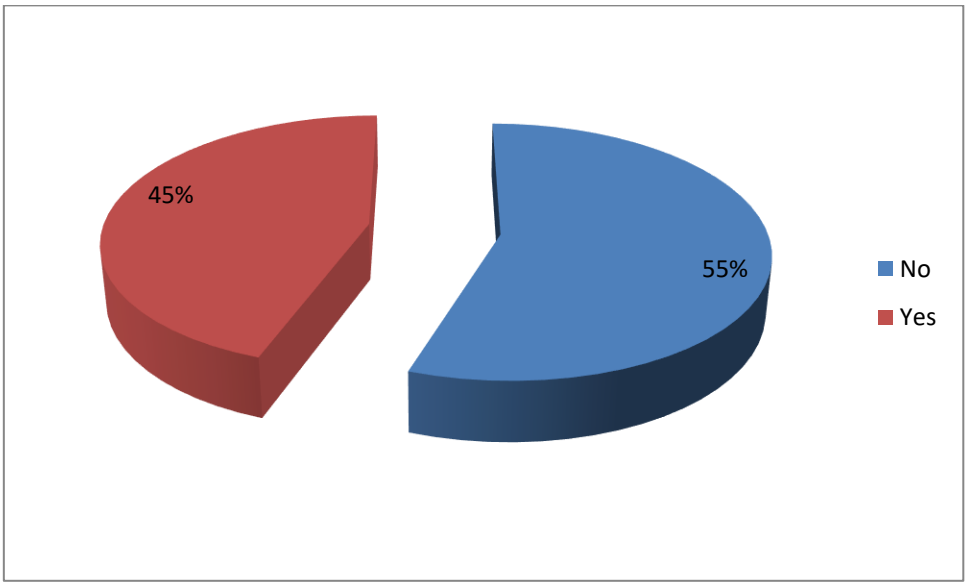


Table 37: Causes of ulcers association with Surgical debridement

	Surgical debridement		
Causes of ulcers	No	Yes	Grand Total
Arterial	9	2	11
Diabetes	5	17	22
Trauma	6	0	6
Venous	16	10	26
Grand Total	36	29	65

In our study 29 patients under went surgical debridement among which the causes of ulcers are diabetic followed by venous and arterial aetiology.

The value of z is 1.274. The value of p is .001. The result is significant at $p < .05$.

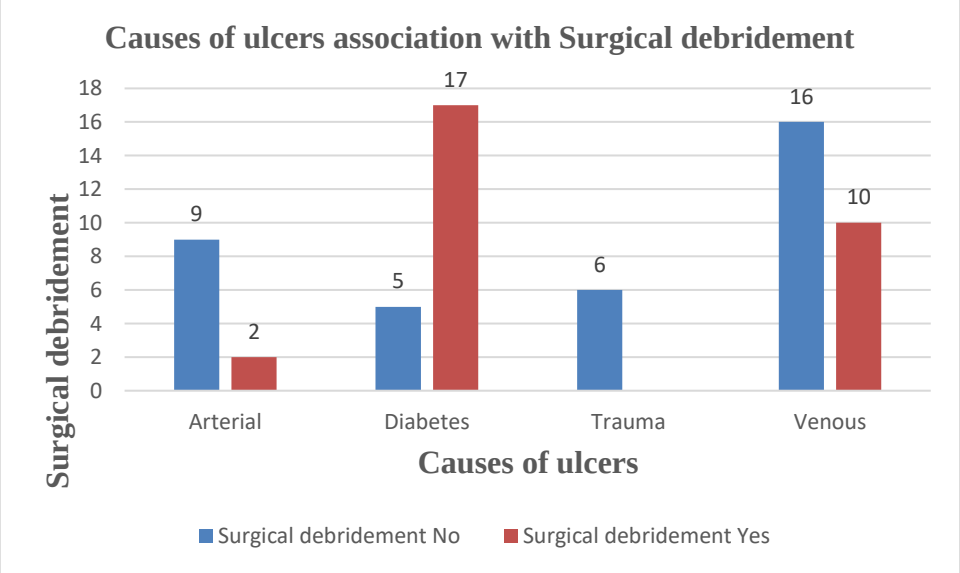


Table 38: Distribution of Skin Grafting

Skin Grafting	Frequency	Percent
No	58	89.2%
Yes	7	10.8%
Total	65	100.0%

In our study, 7 (10.8%) patients had Skin Grafting.

The value of z is 8.946. The value of p is < .00001. The result is significant at p < .05.

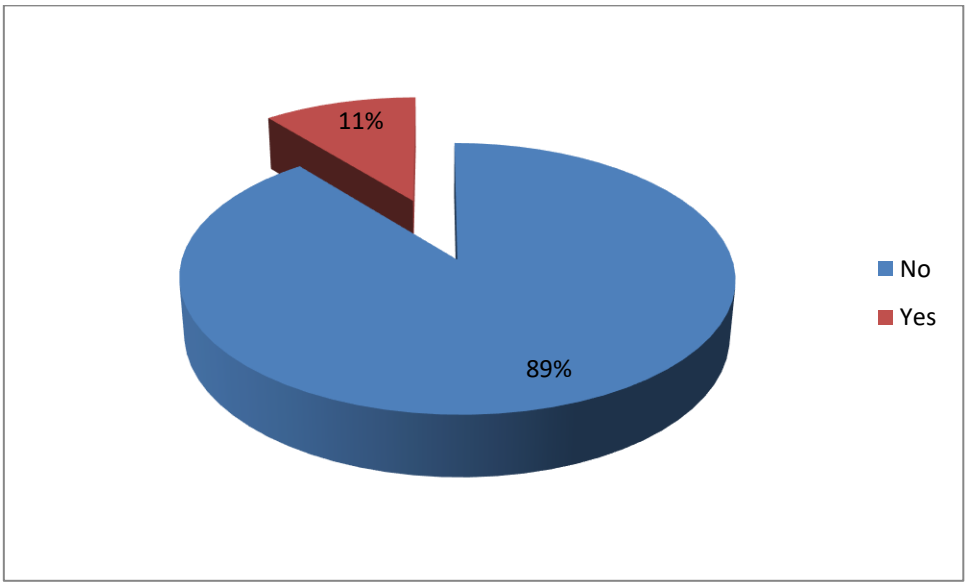


Table 39: Distribution of Subfascial Ligation

Subfascial Ligation	Frequency	Percent
No	63	96.9%
Yes	2	3.1%
Total	65	100.0%

In our study, 2 (3.1%) patients had Subfascial Ligation.

The value of z is 10.7001. The value of p is < .00001. The result is significant at p < .05.

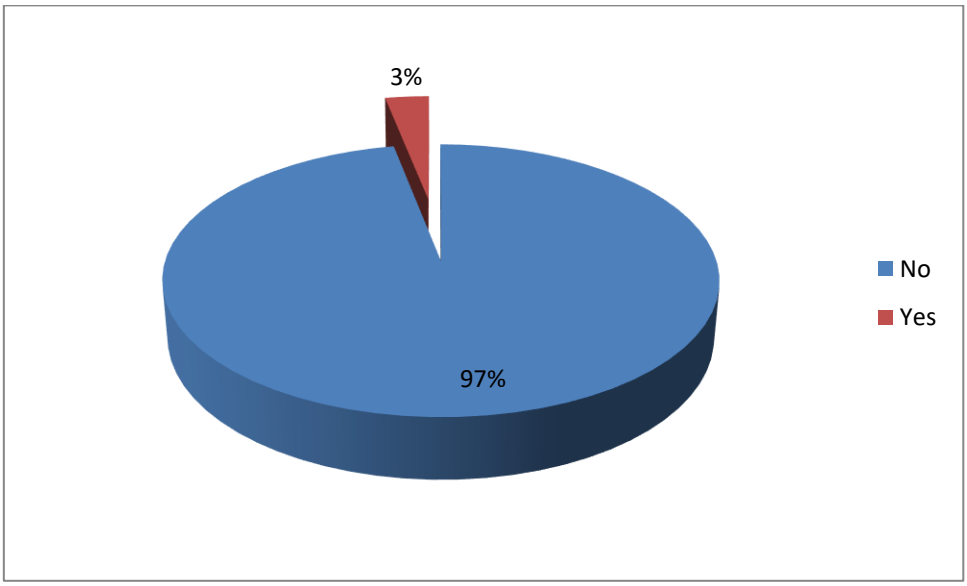


Table 40: Distribution of Ligation and Stripping of Varicose Vein

Ligation And Stripping of Varicose Vein	Frequency	Percent
No	59	90.8%
Yes	6	9.2%
Total	65	100.0%

In our study, 6 (9.2%) patients had Ligation and Stripping of Varicose Vein.

The value of z is 9.2968. The value of p is < .00001. The result is significant at p < .05.

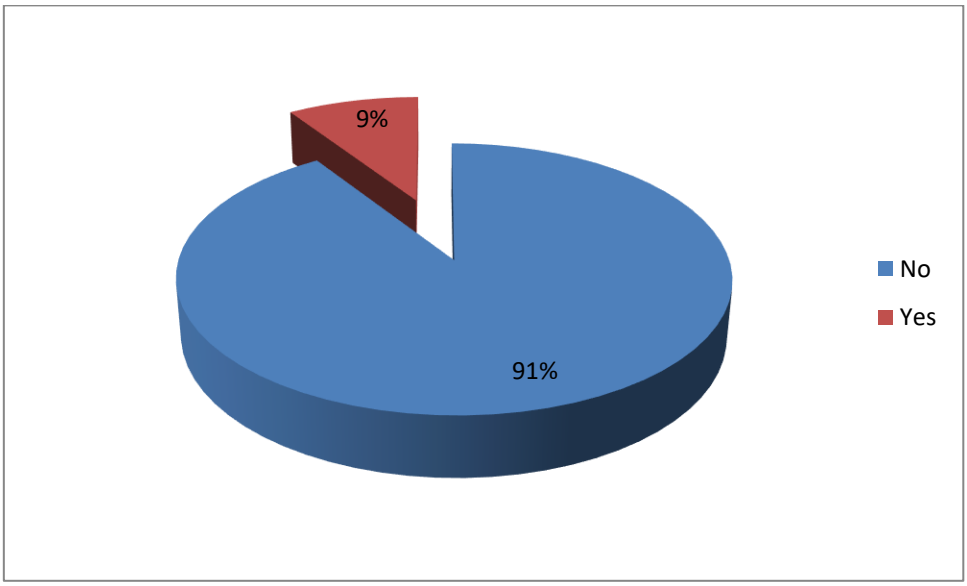


Table 41: Distribution of Wide Excision

Wide Excision	Frequency	Percent
No	64	98.5%
Yes	1	1.5%
Total	65	100.0%

In our study, 1 (1.5%) patient had Wide Excision

The value of z is 11.0509. The value of p is < .00001. The result is significant at p < .05.

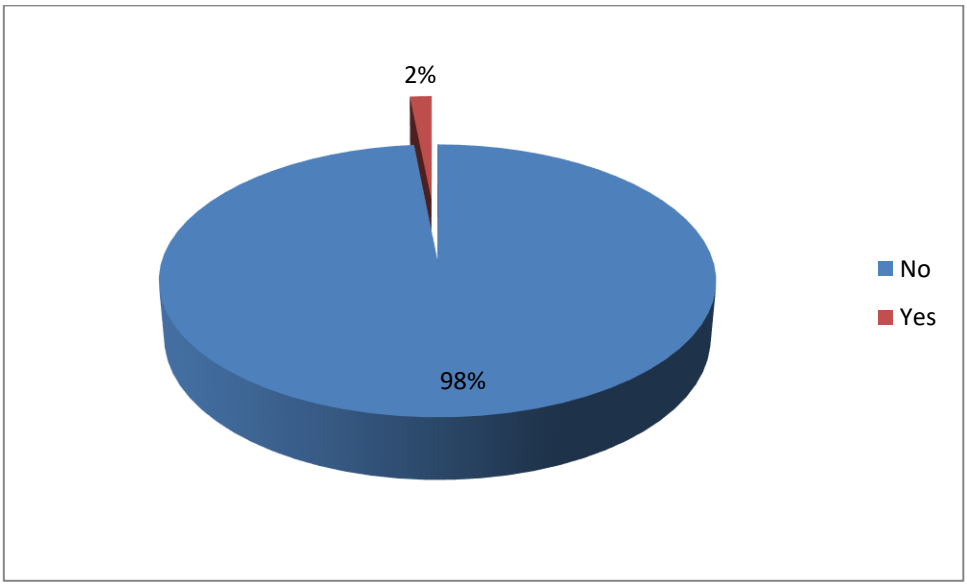


Table 42: Distribution of Lumbar Sympathectomy

Lumbar Sympathectomy	Frequency	Percent
No	63	96.9%
Yes	2	3.1%
Total	65	100.0%

In our study, 2 (3.1%) patients had Lumbar Sympathectomise.

The value of z is 10.7001. The value of p is < .00001. The result is significant at p < .05.

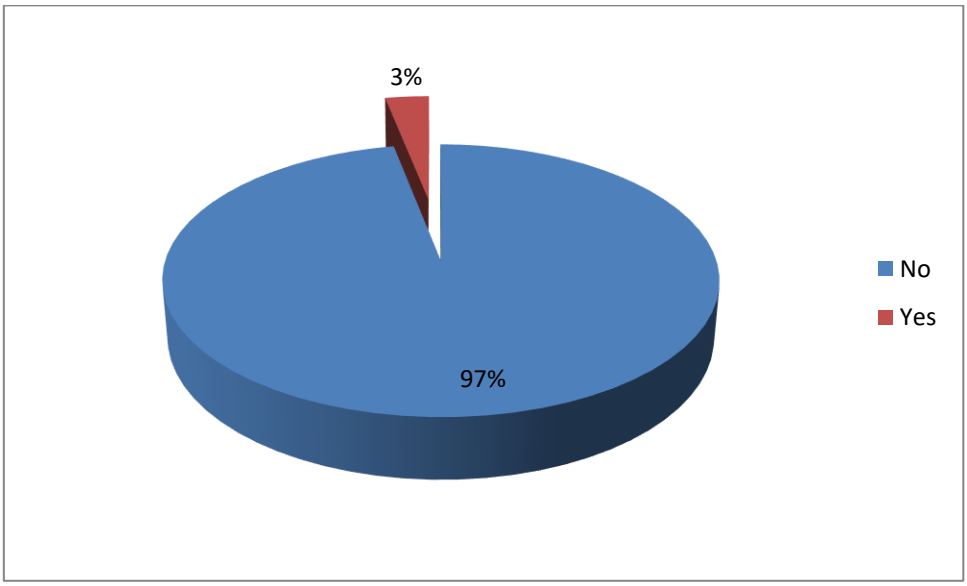


Table 43: Causes of ulcers association with Lumbar sympathectomy

	Lumbar sympathectomy		
Causes of ulcers	No	Yes	Grand Total
Arterial	9	2	11
Diabetes	22	0	22
Trauma	6	0	6
Venous	26	0	26
Grand Total	63	2	65

In our study 2 patients under went lumbar sympathectomy among which the causes of ulcers are of arterial aetiology.

The value of z is 0.927. The value of p is < .001. The result is significant at p < .05.

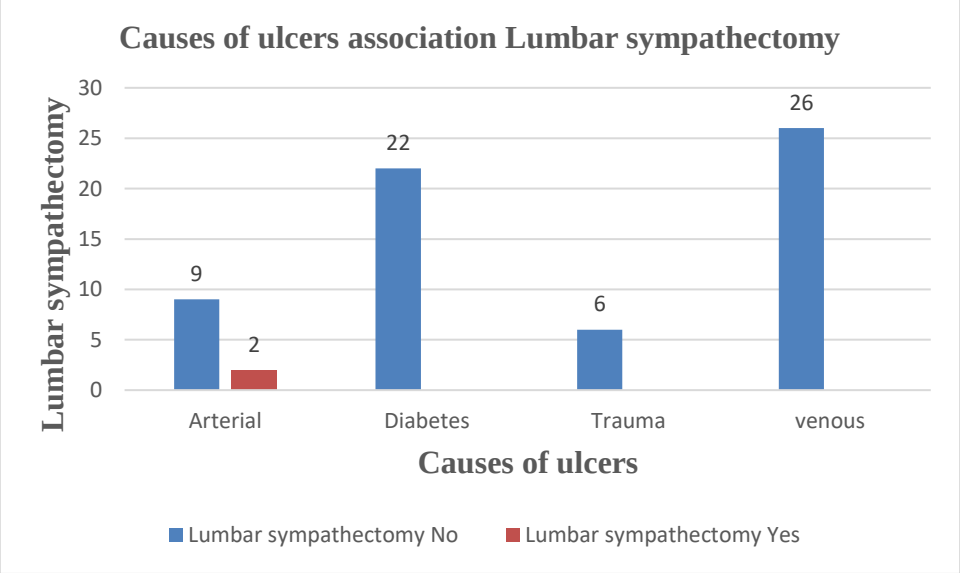


Table 44: Distribution of Amputation

Amputation	Frequency	Percent
No	56	86.2%
Yes	9	13.8%
Total	65	100.0%

In our study, 9 (13.8%) patients had Amputation.

The value of z is 8.2443. The value of p is < .00001. The result is significant at p < .05.

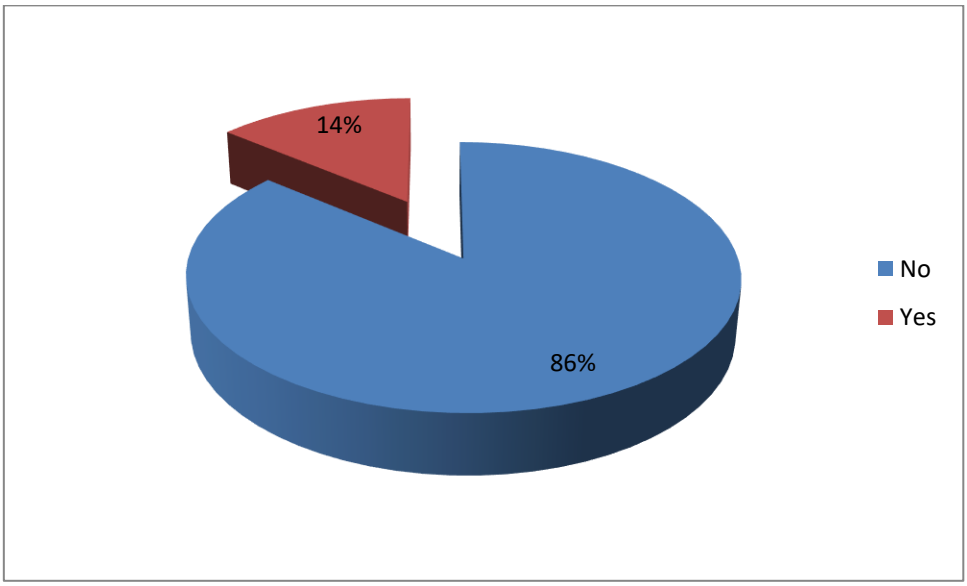


Table 45: Causes of ulcers association with Amputation

	Amputation		
Causes of ulcers	No	Yes	Grand Total
Arterial	4	7	11
Diabetes	21	1	22
Trauma	6	0	6
Venous	25	1	26
Grand Total	56	9	65

In our study 9 patients under went Amputation among them the causes of ulcers are arterial followed by equal number of diabetic and venous aetiology.

The value of z is 0.910. The value of p is < .001. The result is significant at p < .05.

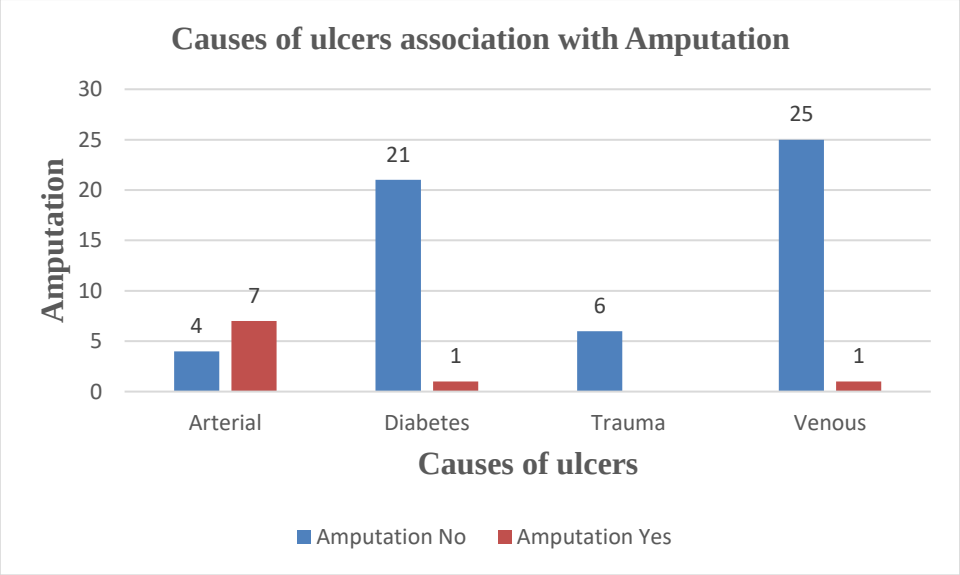


Table 46: Distribution of Follow Up At 15days (Healing)

Follow Up At 15days (Healing)	Frequency	Percent
No	30	46.2%
Yes	35	53.8%
Total	65	100.0%

In our study, 35 (53.8%) patients had Follow Up at 15days (Healing).

The value of z is 0.8771. The value of p is .37886. The result is not significant at p < .05.

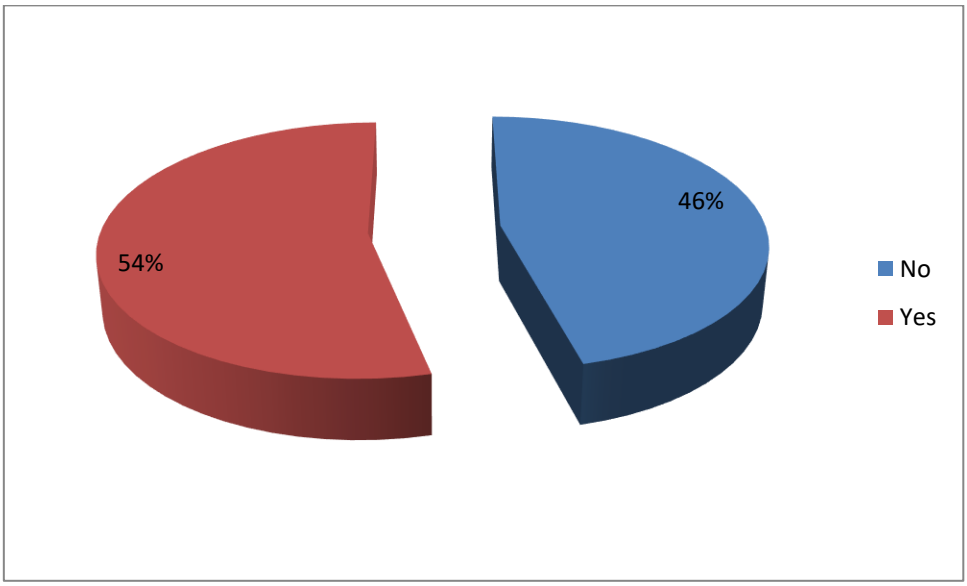


Table 47: Distribution of Follow-up at 2months (Healing)

Follow-up At 2months (Healing)	Frequency	Percent
Yes	65	100.0%
Total	65	100.0%

In our study, 65 (100.0%) patients had follow-up at 2months (Healing).

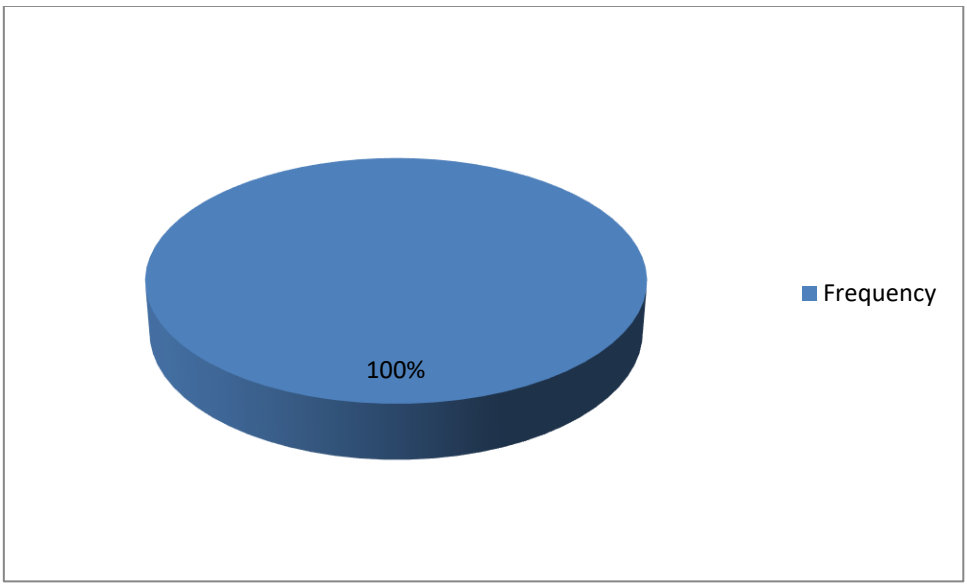
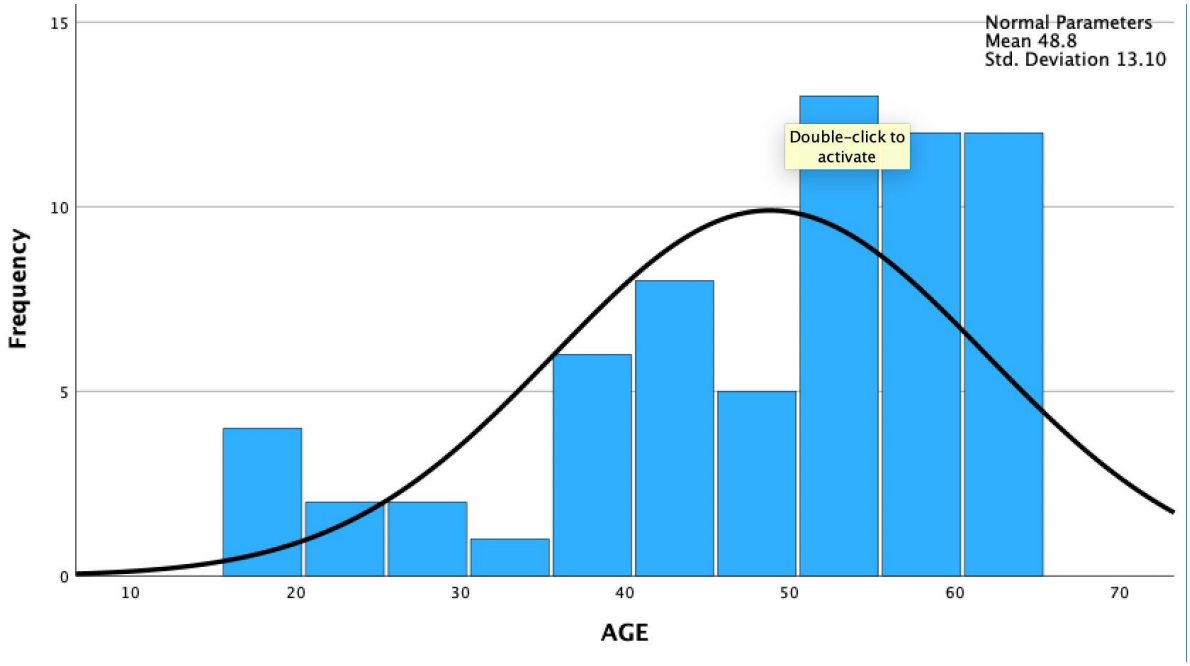


Table 48: Distribution of mean Age

	Number	Mean	SD	Minimum	Maximum	Median
Age	65	48.8000	13.0972	18.0000	65.0000	52.0000

In above table showed that the mean Age (mean±s.d.) of patients was 48.8000± 13.0972.



DISCUSSION AND CONCLUSION

DISCUSSION

The present study was a Prospective interventional study. This study was conducted from July 2021-June 2022 at Department of General Surgery, Santosh Medical College & Hospital Ghaziabad. 65 patients were included in this study.

The prevalence of leg ulcers is probably between 0.18% and 1% (Phillips, Tania et al)[11]. 95% of leg ulcers are due to vascular etiology, (Gilliland)[13] and among all chronic wounds lower extremity venous ulcer dominates the differential diagnosis accounting for up to 90% of the cases (Burton S. Claude)[14], (Callum M. J. et al)[15]. Arterial diseases account for 5% to 10%, most others are due to neuropathy or a combination of both. (Yound J. R) [16]

In our study, out of 65 patients, most of the patients were [25 (38.5%)] 51-60 years of age which was statistically significant (p.00026) (Z=3.6648).

Mohanta PK et al [36] in his study Most of the ulcers were diabetic (36.3%) followed by venous (22.3%) followed by traumatic (15.5%) followed by arterial ulcers (12.4%), and (Z=2.17; p=0.01). Also, according to Yound J. R [16] the distribution of different type of ulcers in different studies varies – 70% to 90% for venous ulcer, 5% to15% for arterial ulcers and 1% to 5% for other ulcers.

In our study ulcer with vascular etiology accounted for only 37 (56.9%) of all ulcers. Out of 65 patients 26 (40.0%) patients had Venous ulcers, 22 (33.8%) patients had Diabetes ulcer, 11 (16.9%) patients had Arterial ulcers, 6 (9.2%) patients had Traumatic ulcers. The value of z is 4.0721. The value of p is < .00001. The result is significant at p < .05.

Mohanta PK et al [36] The mean (mean±SD) age of the patients was 51.94±12.25 years with range 19-80 years and the median age was 53.0 years. Test of proportion showed that the proportion of the patients with age between 40-69 years (84.0%) was significantly higher (Z=9.61; p <0.0001) only 5.1% and 5.2% of patients were with age <30 years and >70 years respectively. Thus, large ulcers were more prevalent in age group 40-69 years.

The study done by Callam M.J [15]. the elderly is not the only population at risk: In his study ulceration began before the age of 40 years in 22% of the population studied.

In our study the mean Age (mean±s.d.) of patients was 48.8000± 13.0972. 8 (12.3%) patients were ≤30 years of age, 7 (10.8%) patients were 31-40 years of age, 13 (20.0%) patient were 41-50 years of age, 25 (38.5%) patients were 51-60 years of age and 12 (18.5%) patients were >60 years of age. The value of z is 3.6648. The value of p is .00026. The result is significant at $p < .05$

In our study out of 8 patients in age group <30 years arterial and venous cause ulcers are most common, out of 7 patients in age group 31-40 years venous cause of ulcers are most common, out of 13 patients in age group 41-50 venous cause of ulcers are most common, out of 25 patients in age group 51-60 years diabetic cause of ulcers are most common followed by venous cause, out of 12 patients in age group > 61 years arterial, venous, diabetic cause of ulcers are equal in number. The value of z is 13.289. The value of p is .001. The result is significant at $p < .05$.

In Callam et al., [37] study a population of about one million, identified 1477 patients with chronic ulcers of the leg. Total 1089 women and 388 men. Women outnumbered men by a ratio of 2.8:1. The median age of the women was 74 and of the men 67. Point prevalence of active leg ulceration was 1.48/1000 of total population. A considerable geographic variation identified evident the range between 1.35-2.09/1000 population.

Mohanta PK et al [36] in his study test of proportion showed that proportion of males 84.5% was significantly higher than that of females 15.5% ($Z=9.89$; $p<0.0001$).

In our study we found that, male population [45 (69.2%)] was higher than the female population [20(30.8%)] which was statistically significant ($p< .00001$) ($Z=4.3853$).

Mohanta PK et al [36] in his study in the left leg (49.2%0 was higher than that of right leg (47.2%) but it was not significant ($Z=0.28$; $p=0.77$). Only 3.6% of the patients were having ulcers in both legs.

In our study, 42 (64.6%) patients had Left Side and 23 (35.4%) patients were Right Side. The value of z is 3.3328. The value of p is .00086. The result is significant at $p < .05$.

Mohanta PK et al [36] in his study in 79.2% of the ulcer's microorganisms were found which was significantly higher than that of no growth (20.8%) (Z=8.25; p<0.0001).

In our study, 35 (53.8%) patients had Growth Seen, 30 (46.2%) patients had Sterile in Culture (Organism). The value of z is 0.8771. The value of p is .37886. The result is not significant at p < .05.

Mohanta PK et al [36] in his study muscle (35.2%) was the most common base of the ulcers followed bone (26.4%) and subcutaneous tissue (24.9%). Bone with tendon and only bone base was found in 6.7% of the cases. (Z=1.58; p=0.11).

In our study, 35 (53.8%) patients had Bone, 15 (23.1%) patients had Muscle and 15 (23.1%) patients had Tendons. The value of z is 3.6056. The value of p is .0003. The result is significant at p < .05.

Mohanta PK et al [36] in his study most of the floors of the ulcers were unhealthy granulation tissue and slough (36.3%) followed by pale granulation tissue and slough (22.3%). Only in 2.1% of the cases it was raised abnormal granulation tissue extending beyond the margin. (Z=2.17; p=0.03).

In our study, 33 (50.8%) patients had Granulation Tissue, 32 (49.2%) patients had Slough in Floor. The value of z is 0.1754. The value of p is .85716. The result is not significant at p < .05.

Mohanta PK et al [36] in his study 55.4% of the ulcers had shallow edge followed by sloping (20.7%) (Z=5.05; p<0.001) and Most of the margin of the ulcers were irregular (38.3%) which was significantly higher (Z=2.46; p=0.0139) followed by thin bluish irregular (22.3%). Only 3.1% of the margin of ulcers was regular.

In our study, 2 (3.1%) patients had averted, 32 (49.2%) patients had Punched Out, 29 (44.6%) patients had Slopping, 2 (3.1%) patients had undermined. The value of z is 5.9871. The value of p is < .00001. The result is significant at p < .05. and in our study, 26 (40.0%) patients had Irregular Margin, 39 (60.0%) patients had Regular Margin. The value of z is 2.2804. The value of p is .0226. The result is significant at p < .05.

As per studies done by Hansson Carita [17] on leg and foot ulcers, ulcers below the line of shoe and feet are considered mostly to be caused by arterial insufficiency and or diabetes. Ulcers on the medial aspect of the ankle in the gaiter zone are mostly caused by venous insufficiency.

Mohanta PK et al [36] in his study most of the patients (61.7%) had ulcer at foot followed by gaiter (19.7%) and leg (18.7%). ($Z=6.04$; $p<0.0001$).

In our study out of 65 patients, 28 (43.1%) patients had Foot, 34 (52.3%) patients had Leg and 3 (4.6%) patients had Thigh in Location of Ulcer. The value of z is 6.0255. The value of p is < .00001. The result is significant at $p < .05$.

In our study, 4 (6.2%) patients had Anterior, 17 (26.2%) patients had Dorsal and 18 (27.2%) patients had Lateral, 18 (27.7%) patients had Lateral, 16 (24.6%) patients had Medial, 5 (7.7%) patients had Plantar, 5 (7.7%) patients had Posterior. The value of z is 3.2747. The value of p is .00108. The result is significant at $p < .05$.

Peripheral vascular diseases increase with age and are 7 times more frequent in60 years old patients when compared to 20 years old. (Hansson Carita) [17]

In our study, arterial and venous diseases were found to be maximum in the age group of 51 to 65 years. And also, venous ulcers were found to be most common in the age group of 51 to 60 years and Arterial were found to be more common in the age group of > 60years years. As most of our patients belong to the working class which involved long hours of standing. In our study, 18 (27.7%) patients had Heavy Work, 14 (21.5%) patients had Moderate and 33 (50.8%) patients had Sedentary Life Style. The value of z is 3.4685. The value of p is .00052. The result is significant at $p < .05$.

Mohanta PK et al [36] in his study venous ulcers were more common in 45-68 years of age (mean 51.58±4.72) with male preponderance (83.7%) and were involved mostly agriculture or service category. Gaiter area of leg involved in 83.7% with ulcer on left leg being more common (76.7%).

Coon et al [38] The prevalence and incidence of venous thromboembolism and other venous disease has been determined as part of a longitudinal study of heal and disease in a Michigan community. An estimated 24 million US citizens have "significant" varicose veins

while 6 to 7 million have stasis changes in the skin of the legs and 400,000 to 500,000 have or have had a varicose ulcer. The relatively high frequency of the second its ions in the adult population.

In our study Out of the 65 patients, 26 (40%) were having venous ulcers out of them 9 (34.6%) patients were in between the age group 51-60years with male preponderance. Most common comorbidities associated are diabetes and hypertension. In out of 26 venous ulcer patients 8 (30.7%) were due to varicose veins. Out of 8 patients with varicose veins, 6 (75%) underwent surgery in form of ligation and or Trendelenburg's operation and 2 (25%) underwent sub fascial ligation which are statistically significant. Remaining venous ulcers mostly managed by surgical debridement. Total 8 (12.3%) patients having Claudication Pain it was statistically significant ($p < .00001$) ($Z=8.5952$).

Mohanta PK et al [36] in his study arterial ulcers were more common in 40–80-year group with male preponderance (91%) because of habit of using tobacco and alcohol,

Out of 65 patients in the study 11 (16.9%) patients having arterial ulcers among them 4 (36.3%) were in the age group more than 60years which was statistically significant with Male preponderance. Raynaud's phenomena seen in 5 (45.4%) patients Itin & Winkelmann [39], and peripheral pulses absent in 5 (45.4%) patients which was statistically significant. Edmonds [40], Most common comorbidies associated with arterial ulcers are hypertension and diabetes. History of smoking is strongly associated with arterial ulcers especially the patient with TAO Mekkes et al.,[41] 2 (18.18%) patients underwent lumbar sympathectomy. Kitahama et al., [49] and 7 (63.63%) patients underwent amputation Emmi et al.,[43] which was statistically significant. In our study 27 patients had HTN in them causes of ulcers are diabetic followed by equal number of arterial and venous etiology. The value of z is 1.028. The value of p is .001. The result is not significant at $p < .05$.

Mohanta PK et al [36] in his study diabetic ulcers were more common around 60 years age group with male preponderance because of habit of using tobacco and alcohol, mostly involving patients engaged in agriculture and business. Majority of the ulcers were located in the foot (88.6%).

In our study Out of the 65 patients, 22 (33.8%) patients having diabetic ulcers among them 12 (54.54%) patients are in the age group 51-to-60-year age group, which was statistically significant. Out of 65 patients in the total 21 (32.3%) having loss of sensations among them 8(38.01%) patients were having diabetic ulcers which was statistically significant. van Deursen, [44], After diabetes, hypertension Singer et al.,[45], is most common co-morbidities associated with diabetic ulcers. Out of total 29 patients who underwent surgical debridement. as management of ulcer 17 (58.62%) which was statistically significant. Falanga,[46]

Cardinal et al.,[47] in his study he retrospectively analyzed the results from two controlled, prospective, randomized pivotal trials of topical wound treatments on 366 chronic venous leg ulcers (VLUs)and 310 diabetic foot ulcers (DFUs) over 12 weeks. VLUs had a significantly higher median wound surface area reduction following clinical visits with surgical debridement as compared with clinical visits with no surgical debridement (34%, p=0.019). Centers where patients were debrided more frequently were associated with higher rates of wound closure in both clinical studies (p=0.007 VLU, p=0.015 DFU) however, there was some minor evidence of a positive benefit of serial debridement in DFUs (odds ratio—2.35, p=0.069).

In our study 6 (9.23%) patients were having traumatic ulcers among them 5 (50%) patients were in the age group 51-60 years age group which was statistically significant. Lower number of patients had [10 (15.4%)] Conservative it was statistically significant (p< .00001) (Z=7.8935).

Skin is the best dressing (Lister). It can be applied as a partial thickness graft or numerous pinch grafts. It is best reserved for large ulcers or those, which will not heal, by conservative management (Gilland E. L., John H. N. Wolf) [13]

In our study, 7 (10.8%) patients had Skin Grafting. The value of z is 8.946. The value of p is < .00001. The result is significant at p < .05.

In our study higher number of patients had [35 (53.8%)] Follow Up at 15days (Healing) it was not statistically significant (p.37886) (Z=0.8771). All patients had Follow-up at 2months (Healing).

Mbunda F et al., [48] descriptive prospective study of patients with chronic lower limb ulcers of 300 patients median age 32 years. The male to female ratio was 2:1. Traumatic ulcer was the most frequent type of ulcer accounting for 60.3% of patients. The leg was commonly affected in 33.7% of cases and the right side (48.7%) was frequently involved. In this study, sixty-eight (22.7%) patients had history of cigarette smoking. Out of 300 patients, 212 (70.7%) had positive aerobic bacterial growth The vast majority of patients, 270 (90.0%) were treated surgically, and surgical debridement was the most common surgical procedure performed in 24.1% of cases. Limb amputation rate was 8.7% Fifty-four (18.0%) patients presented with history of premorbid illness such as diabetes mellitus in 32 (59.3%), chronic pulmonary diseases in 8 (14.8%), hypertension in 6 (11.1%), peripheral vascular diseases in 4 (7.4%) and congenital cardiac diseases and obstructive jaundice in 2 (3.7%). Postoperative complication Predictor (Independent) variables age >20-year P value-0.004, female sex 0.001, pre morbid comorbidities p value0.041, type of ulcers infective p value 0.001, metabolic ulcers p value 0.007.

Kirsner RS et al [49] examined that chronic wounds of the lower extremities are occurring with increasing prevalence. Diagnosis starts with determining whether the wound is one of the four most common chronic wounds: venous leg ulcers, diabetic foot ulcers, pressure ulcers and arterial ulcers. Moreover, despite many recent advances in wound care, the challenge of managing chronic wounds is complicated by the lack of consistently accepted diagnostic methods and wound-care standards. We present a comprehensive yet condensed approach to managing lower-extremity ulcers, from diagnosis to basic management.

Apollonio A et al [50] A large Italian observational multicenter study on vascular ulcers of the lower limbs (Studio Ulcere Vascolari) found that an observational study of 2 years was promoted by the Italian Association for Cutaneous Ulcers (AIUC) in order to monitor the epidemiology of leg ulcers, the trend of healing and the more frequent therapeutic approaches in lower limb ulcers. Fifty-nine sites in 14 different Italian regions involved in the study, with 1333 enrolled patients (1163 patients fully evaluated and followed up for 9 months). A prevalence of females (62%) was observed with a mean age of 70 years and a high rate of hypertension (62%), diabetes (38%) and obesity (29%). Venous ulcer was most frequent (55%), followed by mixed (25%) and diabetic (8·3%) ulcers. These comorbidities were present in different frequencies depending on the type of ulcer, both in the case of hypertension and diabetes ($P < 0{\cdot}001$, $\chi 2$ test) and obesity ($P < 0{\cdot}01$, $\chi 2$ test).

The granulation tissue was very low, with a frequency ranging from around 17% to 23%. The exudate was present in all the ulcers, mainly of medium grade with a frequency that ranged from 41·9% (arterial ulcers) to 47·9% (ulcers combined). it has been found that patients receiving drugs in association with local therapy (LT) showed a significantly (P = 0·001). In venous ulcers, blood hypertension was reported to significantly delay the healing of ulcer with an odds ratio (OR) of 1·45 (CI at 95%: 1·07–2·06; P < 0·02), while obesity showed OR of 1·46 (CI 95%: 1·04–2·04; P = 0·03). Obesity represented a detrimental factor for the healing of mixed ulcers also, with an OR of 1·73 (CI 95%: 1·01–2·97; P < 0·05).

Gokhale Y et al [51] showed that to study the Etiology and Outcomes of Lower Extremity Ulcer in Non- Diabetic Patients. Patients with lower limb ulcers presented with a wide range of pathology. Ulcers due to Vasculitis was the most common etiology (40%) and affected females predominantly (12/16). Venous ulcers were the second most common etiology and predominantly affected men (8/10). It is important to consider differential diagnosis of Vasculitis ulcer in chronic non healing ulcers as they show rapid response to treatment with immunosuppressant. If such ulcers are not promptly diagnosed and treated properly, systemic vasculitis can cause end organ damage or even endanger patient life.

Dhakal P et al [52] This is a retrospective observational study done in patients diagnosed with varicose veins in the great saphenous system Influence of age, gender, duration of illness, and symptoms on pigmentation/ulceration in varicose veins. A total of 223 patient There were a higher number of male patients (male: female=1.47). The mean age was 43.7 years (SD, 13.4); in males, it was 41.8 years (SD, 14.3) and in females, 46.4 years (SD, 11.9). Four variables significantly predicted ulceration in varicose veins. Pain β=0.366, P<0.01), itchiness β=0.355, P<0.01), duration of illness (β=0.204, P<0.01), and male gender (β=0.196, P<0.01) were significant predictors. However, age was not a significant predictor of ulceration.

CONCLUSION

The study findings can be summarized as follows:

Out of 65 patients, most of the patients were 51-60 years age group among them diabetic cause of ulcers was most common followed by venous cause of ulcers. It was found that, the mean Age of patients was [48.8000± 13.0972]. We found that, male population was higher than the female population and male: female ratio was 2.25:1. Our study most of the patients had Sedentary Life Style.

A smaller number of patients had Claudication Pain; a smaller number of patients had Raynaud's Phenomena which was of arterial etiology. We observed that, 44 patients had H/O Smoker among them most common cause of ulcers were diabetic followed by venous and arterial. More number of patients had Bone as Base of ulcer. 11 patients had Arterial System Involvement on USG color doppler among them middle age group and old age group most commonly seen.

Most of the patients had Venous cause of ulcers. Most patients with varicose veins underwent operation i.e., ligation and stripping of veins and or Trendelenburg's operation and sub-fascial ligation following which healing of ulcers seen. patient had Wide Excision who had everted edges of ulcer.

In our study, we have been able to show that leg ulcers are common in elderly population and have negative impact on quality of life of affected patients. It has been found to be more common in males. The highest number of cases was found to be ulcer of the leg associated with ulcer due to venous ulcer, diabetes mellitus, ulcers due to arterial occlusion secondary to atherosclerosis and TAO, traumatic ulcer. A comprehensive assessment of patients' general status, personal habits, skin, vascular status, limb, ulcer characteristics are required to determine the etiology and to formulate effective treatment plan.

Venous ulcers are muscle or subcutaneous tissue deep, with usually shallow edge, usually painful were having thin bluish irregular margin with pale granulation tissue at floor and lipodermatosclerosis, hyperpigmentation and itching in the surrounding area. Venous ulcers were cured by compression stockings, Stripping GSV/SSV and Hook Phlebotomy.

All ulcers were painful involving the foot with atherosclerosis with variable degree of peripheral arterial occlusion being the most common etiopathology. arterial ulcers were found to be deep punched out ulcers with majority had exposed tendon or bone at the base. Majority of arterial ulcers were dry or minimal discharge, containing necrotic tissue with associated hair loss, cold shiny skin, and brittle nail in the surrounding area. Most of the arterial ulcers were

benefited by angiography or angioplasty with or without some sort of amputation.

Diabetic ulcers are mostly commonly seen in old age population with uncontrolled diabetes as comorbid condition which leads the ulcer not to heal quickly. Most of the patient were having loss of sensation due to diabetic neuropathy. History of smoking in them further more decreases the wound healing in them. Along with strict diabetic control and proper antibiotic coverage most of the patients managed with surgical debridement and following which few requires skin grafting after adequate healthy granulation tissues come. Few underwent amputation.

Traumatic ulcers are also most commonly seen in old age people followed by middle age people. Most of the traumatic ulcers were managed conservatively with simple dressings and antibiotic treatment, but few required debridement.

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PROFORMA

Case No: IP No:
Name: DOA:
Age: Sex: DOD:
Address: Occupation:

History

Chief Complaints

History of Presenting Illness

Onset: Duration: Number:
Site: Size : Walking bare foot:
Discharge: Standing long hours:

Past History

Family History

Comorbidities

Examination

General Physical Examination

Pallor Icterus Cyanosis Clubbing Lymphadenopathy Oedema

Systematic Examination CNS: CVS: RS: P/A:

Examination of Ulcers

Inspection:
Site: Number: Size: Shape:
Surrounding area:

Palpation:
Temperature: Tenderness: Floor: Edge:
Margin: Base: Depth:
Discharge: Colour:
Mobility:
Examination Of Regional Lymph Nodes:

Vascular Examination:

Management

Investigations:

Urine R/M: Urine Sugar

Blood Hb%: TC: DC: ESR:

Blood Sugar: RBS: FBS: PPBS: HbA1c:

Urea: Creatinine:

LFT:

Cholesterol: VDRL:

Culture and Sensitivity of pus (if any):

USG/Doppler study:

X-Ray of affected area:

Biopsy:

Treatment

Conservative

Surgical

- Surgical debridement
- Skin grafting
- Subfascial ligation
- Ligation and stripping of varicose veins
- Wide excision
- Lumbar sympathectomy
- Amputation

Follow up at 15days (ulcer healing)
Follow up at 2months (ulcer healing)

SANTOSH MEDICAL COLLEGE AND HOSPITAL (GHAZIABAD)

INFORMED CONSENT (ENGLISH)

I _____ Son/Daughter/Wife of _____ resident of _____hereby declare that I give my full, free and voluntary informed consent in the thesis -" A CLINICOPATHOLOGICAL STUDY OF ULCERS ON THE LOWER LIMB AND ITS MANAGEMENT**".** Dr._____ has
informed me to my full satisfaction, in the language I understand, about the purpose, nature of study and various laboratory investigations for the study to be carried out. I have been informed about the duration of the study and instructions to be followed during the study period.
I give full consent for being enrolled in the above study and I reserve my rights to withdraw from this study, whenever I wish without prejudice of my right to undergo further treatment at Santosh Hospital, Ghaziabad U.P.

Patient's/ Patient's relative's signature Doctor's signature
Or thumb impression

Name: Date:
We have witnessed that the patient signed the above form in the presence of his free will after fully understanding its content.

Signature of Witness

Signature of Investigator
Name: Dr. Shaik Nabi Ruksana
Name Designation: PG Department of General Surgery
Contact No.- 8008116205

INFORMED CONSENT (HINDI)

संतोष मेडिकल कॉलेज एवं अस्पताल (गाजियाबाद)

सहमति पत्र (सूचित)

मैं _____ बेटा / बेटी /पत्नी_____

निवासी _____ घोषणा करता हूं कि
डॉ._____, ने मुझे मेरी भाषा में अपने अध्ययन "**A CLINICOPATHOLOGICAL STUDY OF ULCERS ON THE LOWER LIMB AND ITS MANAGEMENT** " के उद्देश्य के बारे में, और अध्ययन के दौरान अपनाई जाने वाली विभिन्न प्रयोगशाला जांच के बारे में सूचित कर दिया है। मुझे अध्ययनकी अवधि और उसके दौरान अपनाए जाने वाले निर्देश भी मेरी भाषा मे समझा दिए गए हैं| मैं उपरोक्त अध्ययन में बिना किसी दबाव के दाखिला ले रहा हूं और पूर्ण सहमति दे रहा हूं | सन्तोष अस्पताल, गाजियाबाद में अपने आगे के उपचार पर बिना प्रतिकूल प्रभाव डाले इस अध्ययन से और प्रयोगशाला जांच की प्रकृति से मैं जब चाहूँ बाहर जाने के अपने अधिकार को सुरक्षित रख रहा हूं |

रोगी / रोगी के रिश्तेदार के हस्ताक्षर डॉक्टर के हस्ताक्षर

नाम

दिनांक:

मेरी उपस्थिति में मरीज ने पूरी तरह से डाकटर के बताए गए अध्ययन और निर्देशों को समझने के बाद अपनी स्वतंत्र इच्छा से ऊपर के फार्म पर हस्ताक्षर किए हैं ।

हस्ताक्षर नाम: डॉ शेख नबि रुकसाना

पदनाम: पीजी

शल्य चिकित्सा विभाग

संपर्क नंबर- 8008116205

ETHICAL CLEARANCE CERTIFICATE

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(Established u/s 3 of the UGC Act, 1956)

F. No. SU/2021/1830[47] Dated: 17.07.2021

Subject: Grant of Ethical Clearance for Thesis Protocol Topic **"A CLINICOPATHOLOGIC STUDY OF ULCERS ON LOWER LIMB AND ITS MANAGEMENT",** submitted by **Dr. Shaik Nabi Ruksana,** MD/MS student of Batch 2020-2021, Department of **General Surgery**.

*****

With reference to his/her request for grant of Ethical Clearance for the Thesis Protocol entitled **"A CLINICOPATHOLOGIC STUDY OF ULCERS ON LOWER LIMB AND ITS MANAGEMENT"**, **Dr. Shaik Nabi Ruksana** is informed that the Thesis Protocol submitted by him/her was considered by the **Screening Committee** of the Santosh Medical College & Hospital in its meetings held on 19.03.2021, 22.03.2021, 24.03.2021, 25.03.2021, 26.03.2021, 27.03.2021 and 06.04.2021. The recommendations of the **Screening Committee** were considered in detail by the Institutional **Ethics Committee** in its meeting held on **12.07.2021** and the same was **approved** by the **Ethics Committee**.

He /She is informed accordingly for further necessary action.

[Dr. V. P. Gupta]
REGISTRAR

SANTOSH "DEEMED TO BE UNIVERSITY" REGISTRAR GHAZIABAD, NCR DELHI

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2. The Dean, SMC&H
3. The Dean – Research
4. The Dean – Academics
5. The Director - IQAC
6. Dr. Gyanendra Mittal (Guide)
7. HOD of **General Surgery**

PLAGARISM CERTIFICATE

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INTRODUCTION

An ulcer is defined as a disruption of the skin with erosion this breach can also extend further into the contiguous muscle and bone. A chronic ulcer is defined as a full-thickness skin defect with no significant re-epithelization can be observed for more than 4 weeks despite of usual first line of treatment or if the slow healing process could not . The problems of leg ulcer represent a wide spectrum of etiology, pathology, severity and imparts a huge burden of morbidity on the society. The main causes are inefficient or dysfunctional valves of the perforators In extreme rarity, it may be associated with vasculitis During the past three decades considerable break throughs have been seen gaining ever important knowledge Despite all this the treatment options, there is much opportunity for further researches for advances. Despite of having a never-ending list of probable causes, pure venous ulcers and a busket of causes including diabetes, neoplasm l later on in this study. Arterial leg ulcers in short occur due to inadequate blood supply and subsequent tissue hypoperfusion setting off hypoxia. 3 distinct mechanisms could easily be identified all over the world during their lifetime. The etiology of diabetic foot ulcers always has many components. Peripheral neuropathy has been demonstrated again and again to be the main responsible factor to cause of foot ulceration. Evolution of, neuropathy desensitizes the part of the body rendering the patient incompetent to recognized repetitive trauma which is very easily avoided, clearing the road for skin and soft tissue breakdown, allowing the pathogen to enter. Therefore, proper and clear thought about multiple the causes, presentations, and wide range of and subsequent amputation. To add to the woes, an inflammatory environment persisting for a pretty long time, in especially geriatric, chronically diseased or paraplegic or quadriplegic patients. This is also a great concern for the operating surgeons to prevent pressure sores in moribund post operative patients who are usually confined to ICU bed for long time. They usually occur over bony prominences which are very superficial and devoid of much fatty coverings subjected to prolong pressure. They develop due to the compression skin and all the other soft tissues in between a bony prominence and an external surface rendering it sparsely perfused and vulnerable to injury or infections. A detailed and elaborated history may provide many minute and crucial clues to have a idea of probable hen ordering medial malleolus known as Gator's area or less lateral malleolus. Arterial ulcers often affect the toes of lower limb at the earliest, sometimes even impacting the fingers and shin areas in advanced stages. Neuropathic ulcers typically located on the sole of the foot. In regard to lower limb ulcers, Ankle brachial pressure index (ABPI) sheds light to root cause of it. The gold standard and widely available investigation for venous obstruction is the duplex scan which also assesses the location and extent of reflux in venous ulcers, incompetency of perforators and also presence of DVT. It should be combined with USG whole abdomen to rule out any abdominal or pelvic pathology compressing vascularity of the limbs. A lot of emphasis is often given to the where colony count has been mentioned to aid the differentiation between commensal and invading bacteria and should be performed once wound. This is performed or doing a biopsy of the bed of the ulcer. infection is suspected
assessment of the underlying

85% **MATCHING BLOCK 1/1** W

should involve an approach to delivering the correct treatment option for each individual patient,

An ideal management plan for patients with leg ulcers based on the accurate pathophysiology. early strategic and coordinated
AIM AND OBJECTIVES
Aim To study the clinic pathological features of ulcers on the lower limb and its management. Objectives The main objectives of study are as follows:
REVIEW OF LITERATURE
HISTORICAL ASPECT OF LEG ULCERS The mention of chronic leg ulcer could be traced back to very ancient times advocated for he also started described the peculiar ulcers which were associated with varicose veins, could be easily cured by removal of causative dilated veins and he also wrote about post-phlebitic ulcers, rapidly development, always refractory to palliative treatment. Home in 1916 stressed on the importance of Avicenna (982 - 1027 AD) gave a good thorough account of diabetes and was the first person to document and describe diabetic gangrene

BIOSTATISTICS CERTIFICATE

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TO WHOM IT MAY CONCERN

This is to certify that the statistical analysis component of the dissertation titled **"A CLINICO-PATHOLOGICAL STUDY OF ULCER ON THE LOWER LIMB AND ITS MANAGEMENT"** done by Dr. Shaik Nabi Ruksana, 3rd year post graduate resident in the Department of General Surgery, Santosh Medical College and Hospital, Ghaziabad, has been conducted under my direct guidance and supervision.

All the statistical calculations of this study are authentic and correct to the best of my knowledge.

Wish her all the success in her life.

Date: 10/11/2022

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ABBREVIATIONS

SSD	Sliver Sulfadiazine
GSV	Great Saphenous Vein
SSV	Short Saphenous Vein
TAO	Thrombo-angiitis Obliterans
USG	Ultra-Sonography
WLE	Wide Local Excision
IDDM	Insulin Dependent Diabetes Mellitus
NIDDM	Non-Insulin Dependent Diabetes Mellitus
STSG	Split Thickness Skin Graft
FTSG	Full Thickness Skin Graft
BKA	Below Knee Amputation
AKA	Above Knee Amputation
S.NO	SERIAL NUMBER
IP.NO	INPATIENT NUMBER
H/O	HISTORY OF
HTN	HYPERTENSION
DM	DIABETES MELLITUS
HB (gm/dl)	HEMOGLOBIN (gm/dl)
TLC (cu.mm.)	TOTAL LEUKOCYTE COUNT
HBA1C (%)	GLYCATED HEMOGLOBIN
NS	NORMAL STUDY
NAD	NO ABNORMALITY DETECTED
CREATININE (mg)	CREATININE (mg/dl)

MASTER CHART

S.0	IP #	NAME	AGE	AGE GROUPS	GENDER	LIFE STYLE	LOCATION OF ULCER	ASPECT	SIDE	H/O TRAUMA	VARICOSE VEINS	CLAUDICATION PAIN	RAYNAUD'S PHENOMENA	LOSS OF SENSATION	PERIPHERAL PULSES	HTN	DM	H/O SMOKING	EDGES	MARGIN	FLOOR	BASE	SURROUNDING SKIN	DISCHARGE	HB (gm%)	TLC (count	RBS (mg/dl)	HbA1C (%)	CREATININE(mg/dl)	CULTURE (ORGANISM)	USG COLOUR	X-RAY	BIOPSY	CAUSE OF ULCER	CONSERVATIVE	SURGICAL DEBRIDEMENT	SKIN GRAFTING	SKIP AMPUTATION	LIGATION AND	WIDE EXCISION	LUMBAR SYMPATHECTOMY	AMPUTATION	FOLLOW UP AT	FULLOW UP AT
1	17122561	Mr.Bacchu raja	60	51-60	Male	Heavy work	Leg	Medial	Right	0	0	0	0	0	1	0	1	1	Punched out	Irregular	Slough	Bone	1	1	12	14000	212	9	1.54	Growth seen	NS	NAD	NAD	Diabetes	0	1	0	0	0	0	0	0	0	1
2	17123987	Mr.Ram sagar	63	≥61	Male	Heavy work	Foot	Lateral	Left	0	0	0	0	1	1	0	1	1	Punched out	Regular	Slough	Bone	1	1	10	10432	168	7	0.34	Growth seen	NS	NAD	NAD	Diabetes	0	1	0	0	0	0	0	0	0	1
3	17123965	Mrs.Bindu rani	53	51-60	Female	Sedentary	Leg	Medial	Left	0	1	0	0	0	1	1	0	0	Slopping	Regular	Granulation tissue	Bone	1	0	9	8500	101	4.8	0.45	Sterile	Yellow system involved	NAD	NAD	Vellus	0	0	0	0	1	0	0	0	0	1
4	17193853	Mr.Sanjay kumar	41	41-50	Male	Heavy work	Foot	Dorsal	Right	0	0	0	0	0	1	0	0	1	Slopping	Irregular	Granulation tissue	Tendons	0	0	14	8500	78	5.6	0.45	Sterile	NS	NAD	NAD	Vellus	1	0	0	0	0	0	0	0	1	1
5	17877654	Mr.Devender kaur	62	≥61	Female	Sedentary	Foot	Dorsal	Right	0	0	0	0	1	1	0	1	1	Punched out	Regular	Granulation tissue	Tendons	1	1	11	13000	276	6	1.23	Growth seen	NS	NAD	NAD	Diabetes	0	1	0	0	0	0	0	0	1	1
6	17124957	Mr.Dungasi bi	24	≤30	Female	Moderate	Leg	Lateral	Left	1	0	0	0	0	1	0	0	0	Slopping	Regular	Granulation tissue	Muscle	0	0	12	6900	68	4.5	0.24	Sterile	NS	NAD	NAD	Trauma	1	0	0	0	0	0	0	0	1	1
7	17145320	Mrs.Rati devi	65	≥61	Female	Sedentary	Foot	Dorsal	Right	0	0	1	0	1	0	1	1	0	Punched out	Regular	Slough	Bone	1	0	12	8700	133	6	1.04	Growth seen	Arterial system involved	Bone involved	NAD	Arterial	0	0	0	0	0	0	1	0	1	1
8	17153790	Mrs.Vimala devi	46	41-50	Female	Sedentary	Leg	Lateral	Right	1	0	0	0	0	1	1	0	0	Punched out	Regular	Granulation tissue	Bone	0	1	9	4500	79	5	0.23	Sterile	NS	NAD	NAD	Trauma	1	0	0	0	0	0	0	0	0	1
9	17198876	Mr.Dayanand	38	31-40	Male	Moderate	Foot	Dorsal	Left	0	0	0	0	1	1	0	1	1	Punched out	Irregular	Slough	Bone	0	1	8	11165	216	6	1.34	Growth seen	NS	NAD	NAD	Trauma	1	0	0	0	0	0	0	0	1	1
10	17180813	Mrs.Vimala devi	55	51-60	Female	Sedentary	Foot	Dorsal	Left	0	0	0	0	0	1	0	1	1	Punched out	Regular	Granulation tissue	Tendons	1	0	9.3	5290	252	7.2	1.39	Sterile	NS	NAD	NAD	Diabetes	0	0	1	0	0	0	0	0	1	1
11	17145366	Mrs.Bhagwan devi	46	41-50	Female	Sedentary	Leg	Lateral	Left	1	0	0	0	0	1	0	1	0	Slopping	Regular	Slough	Muscle	0	1	11	12000	68	5.8	0.58	Growth seen	NS	NAD	NAD	Vellus	1	0	0	0	0	0	0	0	1	1
12	17144598	Mrs.Noorma	36	31-40	Female	Sedentary	Leg	Medial	Left	0	1	0	0	0	1	0	0	0	Slopping	Regular	Granulation tissue	Bone	1	0	14	4700	92	4.6	0.23	Sterile	Yellow system involved	NAD	NAD	Vellus	0	0	0	0	1	0	0	0	1	1
13	17139035	Mr.Vishwa nthar	62	≥61	Male	Heavy work	Foot	Plantar	Right	0	0	1	1	1	1	0	1	1	Punched out	Regular	Slough	Bone	0	0	7.6	25460	92	6.2	1.42	Growth seen	Arterial system involved	Bone involved	NAD	Arterial	0	0	0	0	0	0	1	0	1	1
14	17150510	Mr.Sumdar sharma	60	51-60	Male	Heavy work	Foot	Plantar	Right	0	0	0	0	1	1	1	1	1	Slopping	Regular	Granulation tissue	Tendons	0	0	9.2	13900	101	8.2	1.3	Growth seen	NS	NAD	NAD	Diabetes	0	1	0	0	0	0	0	0	1	1
15	17151494	Mr.Sanjay	23	≤30	Male	Sedentary	Leg	Anterior	Left	0	0	0	0	0	1	1	0	1	Punched out	Regular	Slough	Bone	0	0	8	8900	89	5.7	0.89	Sterile	Arterial system involved	NAD	NAD	Arterial	0	1	0	0	0	0	0	0	1	1
16	17154340	Mr.Sachin	37	31-40	Male	Sedentary	Leg	Lateral	Left	1	0	0	0	0	1	0	0	1	Slopping	Irregular	Slough	Muscle	0	0	15	13000	83	5	0.23	Sterile	NS	NAD	NAD	Trauma	1	0	0	0	0	0	0	0	1	1
17	17199887	Ms.Pallavi	19	≤30	Female	Moderate	Thigh	Lateral	Right	1	0	0	0	0	1	0	0	0	Slopping	Regular	Granulation tissue	Muscle	1	1	12	16000	65	4.2	0.34	Growth seen	Yellow system involved	NAD	NAD	Vellus	0	1	0	0	0	0	0	0	0	1
18	17198333	Mr.Nitu yadav	18	≤30	Male	Moderate	Foot	Lateral	Left	0	0	0	0	0	1	1	0	1	Punched out	Regular	Slough	Bone	0	0	11	7600	87	4.2	0.43	Sterile	Arterial system involved	NAD	NAD	Arterial	0	1	0	0	0	0	0	0	1	1
19	17218926	Mrs.Bobbi gupta	58	51-60	Female	Sedentary	Thigh	Lateral	Right	1	0	0	0	0	1	1	1	0	Slopping	Regular	Granulation tissue	Muscle	0	1	8	14000	102	5	1.34	Growth seen	NS	NAD	NAD	Vellus	0	1	0	0	0	0	0	0	0	1
20	17225997	Mr.Bhushan	52	51-60	Male	Sedentary	Foot	Dorsal	Left	0	0	1	1	1	0	0	1	1	Punched out	Regular	Slough	Bone	0	1	7	24000	120	4.6	2.45	Growth seen	Arterial system involved	Bone involved	NAD	Arterial	0	0	0	0	0	0	1	0	1	1
21	17164351	Mr.Madan pal	59	51-60	Male	Sedentary	Foot	Dorsal	Right	0	0	0	0	0	1	0	1	1	Punched out	Irregular	Granulation tissue	Tendons	1	1	12	9800	132	8	1.2	Sterile	NS	NAD	NAD	Diabetes	0	1	1	0	0	0	0	0	1	1
22	17161245	Mrs.Gyanwati	54	51-60	Female	Sedentary	Leg	Medial	Right	0	1	0	0	1	1	0	1	0	Slopping	Irregular	Granulation tissue	Bone	1	1	10	7600	56	3.4	0.12	Sterile	Yellow system involved	NAD	NAD	Vellus	0	0	0	0	1	0	0	0	1	1
23	17104332	Mr.Niyaz ahmed	54	51-60	Male	Sedentary	Foot	Posterior	Left	0	0	0	0	1	1	0	1	1	Punched out	Regular	Slough	Bone	0	0	9.2	8200	83	6	0.15	Sterile	Yellow system involved	NAD	NAD	Vellus	0	1	0	0	0	0	0	0	0	1
24	17194433	Mr.Shiv kumar	47	41-50	Male	Heavy work	Leg	Medial	Right	0	0	0	0	0	1	0	1	1	Slopping	Irregular	Granulation tissue	Bone	1	1	12	6700	98	5.4	1.34	Growth seen	Yellow system involved	NAD	NAD	Vellus	0	1	0	0	0	0	0	0	0	1
25	17145432	Mrs.Ramwati	61	≥61	Female	Sedentary	Foot	Dorsal	Right	1	0	0	0	0	1	1	0	0	Everted	Irregular	Granulation tissue	Bone	1	0	10	7600	92	6	1.22	Sterile	NS	NAD	Premalignant growth	Vellus	0	0	0	0	1	0	0	0	0	1
26	17135433	Mr.Shalendra	30	≤30	Male	Sedentary	Foot	Dorsal	Left	0	0	0	0	0	1	0	0	1	Punched out	Regular	Slough	Bone	1	0	12	6430	72	5.2	0.23	Growth seen	Arterial system involved	Bone involved	NAD	Arterial	0	0	0	0	0	0	1	0	1	1
27	17137892	Mrs.Kalawati	45	41-50	Female	Moderate	Leg	Anterior	Left	1	0	0	0	0	1	1	0	0	Punched out	Regular	Slough	Bone	0	1	14	7800	65	6	0.54	Sterile	Yellow system involved	NAD	NAD	Vellus	0	1	0	0	0	0	0	0	1	1
28	17137363	Mr.Ashok	52	51-60	Male	Sedentary	Foot	Dorsal	Left	1	0	0	0	0	1	1	1	1	Everted	Irregular	Slough	Bone	1	1	8.8	28660	210	9.8	1.51	Growth seen	NS	Bone involved	Premalignant growth	Diabetes	0	0	0	0	0	0	1	0	1	1
29	17162662	Mr.Zameer ahmed	65	≥61	Male	Heavy work	Foot	Dorsal	Right	0	0	0	0	0	1	1	0	1	0	Slopping	Regular	Granulation tissue	Tendons	1	0	12	7900	156	8.25	0.23	Sterile	NS	NAD	NAD	Diabetes	0	1	0	0	0	0	0	0	1	1
30	17157832	Mr.Tarachand	65	≥61	Male	Moderate	Foot	Posterior	Left	0	0	0	0	1	1	1	1	1	punched out	Regular	Slough	Bone	0	0	7.8	14000	143	6	0.21	Growth seen	NS	NAD	NAD	Diabetes	0	1	0	0	0	0	0	0	0	1
31	17159254	Mrs.Afsana	45	41-50	Female	Moderate	Foot	Dorsal	Left	1	0	0	0	0	1	0	1	1	Slopping	Irregular	Slough	Tendons	1	1	11	12000	98	5	0.12	Growth seen	Yellow system involved	NAD	NAD	Vellus	0	1	0	0	0	0	0	0	1	1
32	17183236	Mrs.Rani	42	41-50	Female	Moderate	Foot	Medial	Right	0	0	0	0	0	1	0	1	0	Slopping	Irregular	Granulation tissue	Tendons	0	0	9.2	11670	167	7	1.23	Growth seen	NS	NAD	NAD	Diabetes	0	1	0	0	0	0	0	0	1	1
33	17160559	Mr.Zahid alam	65	≥61	Male	Sedentary	Leg	Medial	Left	0	1	0	0	0	1	1	1	1	Punched out	Irregular	Slough	Bone	1	1	11.2	9400	78	5.9	0.67	Growth seen	Yellow system involved	NAD	NAD	Vellus	0	0	0	0	1	0	0	0	0	1
34	17166365	Mr.Dayanand	48	41-50	Male	Moderate	Foot	Dorsal	Right	0	0	1	1	0	0	1	0	1	Punched out	Regular	Slough	Bone	0	0	9	17440	81	6	1.12	Growth seen	Arterial system involved	Bone involved	NAD	Arterial	0	0	0	0	0	0	1	0	1	1
35	17172012	Mr.Hari chand	59	51-60	Male	Heavy work	Leg	Lateral	Left	1	0	0	0	0	1	0	0	1	Slopping	Irregular	Granulation tissue	Muscle	0	0	13	8730	71	4.7	0.59	Sterile	NS	NAD	NAD	Trauma	0	0	1	0	0	0	0	0	1	1
36	17144093	Mr.Rakesh	18	≤30	Male	Moderate	Leg	Posterior	Left	1	0	0	0	0	1	0	1	1	Undermined	Regular	Granulation tissue	Tendons	1	0	10	7200	63	4.9	0.34	Growth seen	NS	NAD	NAD	Diabetes	0	1	0	0	0	0	0	0	0	1
37	17138245	Mr.Saswata	55	51-60	Male	Sedentary	Leg	Anterior	Left	0	0	0	0	0	1	1	1	1	Punched out	Regular	Granulation tissue	Tendons	0	0	9.8	4500	169	7.4	0.98	Sterile	NS	NAD	NAD	Diabetes	1	0	0	0	0	0	0	0	1	1
38	17138765	Mr.Ashwani kumar	44	41-50	Male	Sedentary	Foot	Dorsal	Left	0	0	1	1	1	1	0	1	1	punched out	Regular	Slough	Bone	0	0	12.8	13000	88	6.1	1.43	Growth seen	Arterial system involved	NAD	NAD	Arterial	0	0	0	0	0	0	1	0	1	1
39	17138233	Mr.Ram bhavan	55	51-60	Male	Heavy work	Leg	Lateral	Left	1	0	0	0	0	1	0	0	1	Slopping	Irregular	Granulation tissue	Muscle	0	0	14	8400	84	5.7	0.65	Sterile	NS	NAD	NAD	Trauma	0	0	1	0	0	0	0	0	1	1
40	17138798	Mr.Faiz muhammad	58	51-60	Male	Heavy work	Leg	Lateral	Right	0	0	0	0	0	0	0	0	0	Slopping	Regular	Granulation tissue	Bone	0	0	9	9700	78	5.2	0.29	Sterile	NS	NAD	NAD	Vellus	1	0	0	0	0	0	0	0	1	1
41	17149834	Mr.Rajendra	30	≤30	Male	Heavy work	Leg	Medial	Left	0	0	0	0	0	1	1	1	1	Punched out	Irregular	Slough	Bone	1	1	11	10560	82	5.9	0.56	Growth seen	Yellow system involved	NAD	NAD	Vellus	0	1	0	0	0	0	0	0	1	1
42	17148123	Mr.Tirchanigaur	59	51-60	Male	Sedentary	Leg	Medial	Right	0	1	0	0	0	1	1	0	1	Slopping	Regular	Granulation tissue	Bone	1	1	8.4	11020	96	4.8	1.06	Growth seen	Yellow system involved	NAD	NAD	Vellus	0	0	0	1	0	0	0	0	0	1
43	17165218	Mr.Praveen	37	31-40	Male	Sedentary	Leg	Posterior	Left	0	0	0	0	0	1	0	0	1	Punched out	Regular	Granulation tissue	Muscle	0	0	13	10040	84	5.2	0.68	Sterile	NS	NAD	NAD	Vellus	0	0	1	0	0	0	0	0	1	1
44	17140145	Mr.Avdesh	52	51-60	Male	Heavy work	Thigh	Lateral	Left	0	0	0	0	0	1	0	1	1	Punched out	Irregular	Slough	Muscle	1	1	7.8	16020	265	7.4	1.87	Growth seen	NS	NAD	NAD	Diabetes	0	1	0	0	0	0	0	0	0	1
45	17149567	Mrs.Mamta	47	41-50	Female	Moderate	Leg	Lateral	Left	0	0	0	0	0	1	1	1	0	Slopping	Regular	Granulation tissue	Muscle	0	0	10.2	9600	102	5.5	0.69	Sterile	NS	NAD	NAD	Diabetes	0	1	0	0	0	0	0	0	1	1
46	17140789	Mr.Shaheer	38	31-40	Male	Heavy work	Leg	Medial	Right	1	0	0	0	0	1	0	0	1	Slopping	Regular	Slough	Tendons	1	1	8.6	11640	64	6	0.63	Growth seen	Yellow system involved	NAD	NAD	Vellus	0	1	0	0	0	0	0	0	0	1
47	17150123	Mr.Raj kumar	34	31-40	Male	Sedentary	Leg	Medial	Left	0	0	1	1	1	0	1	0	1	Punched out	Regular	Slough	Bone	0	0	7	9830	79	5.1	0.44	Sterile	Arterial system involved	NAD	NAD	Arterial	0	0	0	0	1	0	0	0	1	1
48	17150254	Mrs.Tara devi	55	51-60	Female	Sedentary	Leg	Anterior	Left	1	0	0	0	0	1	1	1	1	Slopping	Irregular	Slough	Bone	0	0	12	12010	107	6.1	0.83	Growth seen	NS	NAD	NAD	Diabetes	0	1	0	0	0	0	0	0	1	1
49	17150456	Mrs.Kusum devi	59	51-60	Female	Moderate	Foot	Posterior	Left	0	0	0	0	1	1	1	0	0	Punched out	Regular	Slough	Bone	0	0	10.2	7840	104	4.9	1.05	Growth seen	NS	NAD	NAD	Vellus	0	1	0	0	0	0	0	0	0	1
50	17150384	Mr.Vikas kumar	58	51-60	Male	Sedentary	Leg	Medial	Right	0	1	0	0	0	1	0	1	1	Slopping	Irregular	Granulation tissue	Bone	1	1	14	9200	82	5.3	0.68	Sterile	Yellow system involved	NAD	NAD	Vellus	0	0	0	0	1	0	0	0	0	1
51	17150783	Mrs.Samanta devi	45	41-50	Female	Sedentary	Leg	Lateral	Left	1	0	0	0	0	1	0	1	0	Slopping	Irregular	Granulation tissue	Muscle	0	0	11	7560	93	5.9	1.07	Sterile	NS	NAD	NAD	Diabetes	0	0	1	0	0	0	0	0	1	1
52	17160124	Mr.Varun tyagi	37	31-40	Male	Sedentary	Foot	Plantar	Left	0	0	0	0	0	1	1	1	1	Undermined	Regular	Granulation tissue	Muscle	0	1	7	3800	128	6.8	1.21	Growth seen	NS	NAD	NAD	Diabetes	0	1	0	0	0	0	0	0	1	1
53	17103954	Mr.Sonu	19	≤30	Male	Moderate	Leg	Lateral	Left	0	0	0	0	0	1	0	0	0	Slopping	Regular	Granulation tissue	Tendons	0	0	10	6270	89	5.2	1.08	Sterile	NS	NAD	NAD	Vellus	1	0	0	0	0	0	0	0	1	1
54	17113472	Mr.Ramawatpal	65	≥61	Male	Sedentary	Leg	Medial	Left	0	0	0	0	0	1	1	0	1	Slopping	Irregular	Granulation tissue	Bone	1	1	12	10000	104	6	1.18	Sterile	Yellow system involved	NAD	NAD	Vellus	0	1	0	0	0	0	0	0	1	1
55	17111584	Mr.Suresh chand	53	51-60	Male	Sedentary	Foot	Dorsal	Left	0	0	0	0	0	1	1	1	0	Slopping	Irregular	Granulation tissue	Tendons	0	0	13	7600	145	7.8	0.85	Sterile	NS	NAD	NAD	Diabetes	0	0	1	0	0	0	0	0	1	1
56	17084781	Mr.Rohtash	58	51-60	Male	Heavy work	Foot	Plantar	Right	0	0	0	0	0	1	0	0	0	Slopping	Regular	Slough	Muscle	0	0	14	5600	108	5	0.72	Sterile	NS	NAD	NAD	Vellus	1	0	0	0	0	0	0	0	1	1
57	17176285	Mr.Chanan singh	65	≥61	Male	Heavy work	Leg	Lateral	Left	0	0	1	0	1	1	1	1	1	Punched out	Regular	Slough	Bone	0	0	8	17000	128	4.8	2	Growth seen	Arterial system involved	NAD	NAD	Arterial	0	0	0	0	0	0	1	0	1	1
58	17169993	Mr.Lakhmi chand	42	41-50	Male	Moderate	Leg	Medial	Right	0	0	0	0	1	1	0	1	1	Punched out	Irregular	Slough	Bone	1	1	11	9800	68	5.8	1.06	Growth seen	Yellow system involved	NAD	NAD	Vellus	0	0	0	0	0	0	1	0	1	1
59	17169968	Mr.Bunty saini	56	51-60	Male	Sedentary	Foot	Plantar	Right	0	0	0	0	0	1	0	1	1	Punched out	Regular	Granulation tissue	Muscle	0	0	12	10050	78	5.2	0.21	Growth seen	NS	NAD	NAD	Diabetes	0	1	0	0	0	0	0	0	1	1
60	17113454	Mr.Saswata	52	51-60	Male	Heavy work	Foot	Dorsal	Left	0	0	0	0	0	1	1	1	1	Punched out	Irregular	Slough	Tendons	1	1	11	14000	109	7	1.1	Growth seen	NS	NAD	NAD	Diabetes	0	1	0	0	0	0	0	0	1	1
61	17163588	Mr.Vinay kumar	61	≥61	Male	Heavy work	Leg	Medial	Right	0	1	0	0	0	1	0	0	1	Slopping	Irregular	Granulation tissue	Bone	1	0	12	10000	114	4.8	0.12	Sterile	Yellow system involved	NAD	NAD	Vellus	0	0	0	0	1	0	0	0	0	1
62	17188524	Mr.Bharat	56	51-60	Male	Sedentary	Leg	Lateral	Left	1	0	0	0	0	1	1	0	1	Slopping	Regular	Granulation tissue	Muscle	0	0	13	12000	105	5	0.98	Growth seen	NS	NAD	NAD	Trauma	1	0	0	0	0	0	0	0	1	1
63	17188876	Mr.Anil	42	41-50	Male	Sedentary	Leg	Medial	Left	0	1	0	0	1	1	0	1	0	Punched out	Irregular	Slough	Bone	1	1	9	9800	76	5.4	0.78	Growth seen	Yellow system involved	NAD	NAD	Vellus	0	0	0	1	0	0	0	0	0	1
64	17188528	Mr.Deva	62	≥61	Male	Heavy work	Foot	Dorsal	Left	0	0	1	0	1	0	1	1	1	Punched out	Regular	Slough	Bone	0	0	9.8	15000	107	6	0.65	Growth seen	Arterial system involved	NAD	NAD	Arterial	0	0	0	0	0	0	1	0	1	1
65	17174767	Mr.Shalini	53	51-60	Female	Sedentary	Leg	Lateral	Left	1	0	0	0	1	1	0	1	1	Punched out	Irregular	Slough	Tendons	1	1	11	7800	112	6.1	0.92	Sterile	NS	NAD	NAD	Diabetes	0	1	0	0	0	0	0	0	1	1